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s

Set     Items   Description
S1          1   AU='NEWELL MARTHA K'
S2          0   S1 AND (CD40L OR CD40 OR CD40(W)LIGAND)
S3       1207   (CD40L OR CD40 OR CD40(W)LIGAND) AND (TCR OR T(W)CELL(W)RE-
            CEPTOR? OR REARRANGEMENT?)
S4        413   (CD40L OR CD40 OR CD40(W)LIGAND) (20N) (TCR OR T(W)CELL(W)-
            RECEPTOR? OR REARRANGEMENT?)
S5        219   RD S4 (unique items)
S6         47   S5 AND INDUC?(10N)(TCR OR T(W)CELL(W)RECEPTOR? OR REARRANG-
            EMENT)
S7         47   RD S6 (unique items)
? s (multimer? or oligomer?)(10n)(Cd40L or cd40(w)ligand) and (tcr or
t(W)cell(W)receptor? or rearrangement)
Processing
Processing
          18350  MULTIMER?
         100114  OLIGOMER?
           5037  CD40L
          20273  CD40
         374036  LIGAND
           9336  CD40(W)LIGAND
             27  (MULTIMER? OR OLIGOMER?)(10N)(CD40L OR CD40(W)LIGAND)
          61485  TCR
        4717901  T
        8430791  CELL
        2553258  RECEPTOR?
          67997  T(W)CELL(W)RECEPTOR?
         150184  REARRANGEMENT
      S8      0  (MULTIMER? OR OLIGOMER?)(10N)(CD40L OR CD40(W)LIGAND) AND
                 (TCR OR T(W)CELL(W)RECEPTOR? OR REARRANGEMENT)
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09/470494

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b 410
       02mar04 16:18:29 User208760 Session D2442.1
            $0.31    0.089 DialUnits File1
     $0.31  Estimated cost File1
     $0.31  Estimated cost this search
     $0.31  Estimated total session cost   0.089 DialUnits

File 410:Chronolog(R)  1981-2004/Feb
       (c) 2004 The Dialog Corporation

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? set hi ;set hi
HILIGHT set on as ''
HILIGHT set on as ''
? begin 5,73,155,399
       02mar04 16:18:37 User208760 Session D2442.2
            $0.00    0.072 DialUnits File410
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     $0.03  TELNET
     $0.03  Estimated cost this search
     $0.34  Estimated total session cost   0.161 DialUnits

SYSTEM:OS  - DIALOG OneSearch
  File   5:Biosis Previews(R)  1969-2004/Feb W4
         (c) 2004 BIOSIS
  File  73:EMBASE  1974-2004/Feb W4
         (c) 2004 Elsevier Science B.V.
  File 155:MEDLINE(R)  1966-2004/Feb W4
         (c) format only 2004 The Dialog Corp.
*File 155: Medline has been reloaded. Accession numbers
have changed. Please see HELP NEWS 154 for details.
  File 399:CA SEARCH(R)  1967-2004/UD=14010
         (c) 2004 American Chemical Society
*File 399: Use is subject to the terms of your user/customer agreement.
Alert feature enhanced for multiple files, etc. See HELP ALERT.

      Set  Items  Description
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? e au=newell martha ?

Ref    Items  Index-term
E1         1  AU=NEWELL MARK A
E2         1  AU=NEWELL MARK E
E3         0 *AU=NEWELL MARTHA ?
E4         1  AU=NEWELL MARTHA K
E5         2  AU=NEWELL MELVIN A
E6         1  AU=NEWELL MICHAEL J
E7         1  AU=NEWELL MIKE
E8         2  AU=NEWELL MORRIS L.L.
E9        20  AU=NEWELL N
E10       29  AU=NEWELL N D
E11        2  AU=NEWELL N E
E12        1  AU=NEWELL N S

       Enter P or PAGE for more
? s e4
      S1       1  AU='NEWELL MARTHA K'
? t s1/3/all

 1/3/1     (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
(c) 2004 BIOSIS. All rts. reserv.
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0014376959 BIOSIS NO.: 200300345678
Compositions and methods for promoting wound healing
AUTHOR: **Newell Martha K** (Reprint); Askill Ian; Pierce Javin; Crisp William
AUTHOR ADDRESS: Colorado Springs, CO, USA**USA
JOURNAL: Official Gazette of the United States Patent and Trademark Office Patents 1271 (4): June 24, 2003 2003
MEDIUM: e-file
PATENT NUMBER: US 6582713 PATENT DATE GRANTED: June 24, 2003 20030624
PATENT CLASSIFICATION: 424-407 PATENT ASSIGNEE: Univ. of Colorado - Colorado Springs, Colorado Springs, CO, USA; Medlogic Global Corp., Devon, UK; Anodose Dermal Product, Inc., Stowe, VT, USA PATENT COUNTRY: USA
ISSN: 0098-1133 _(ISSN print)
DOCUMENT TYPE: Patent
RECORD TYPE: Abstract
LANGUAGE: English

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? s s1 and (cd40L or cd40 or cd40(w)ligand)
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            5037  CD40L
           20273  CD40
           20273  CD40
          374036  LIGAND
            9336  CD40(W)LIGAND
      S2       0  S1 AND (CD40L OR CD40 OR CD40(W)LIGAND)
? s (cd40L or cd40 or cd40(w)ligand) and (tcr or t(w)cell(w)receptor? or
rearrangement?)
Processing
Processing
            5037  CD40L
           20273  CD40
           20273  CD40
          374036  LIGAND
            9336  CD40(W)LIGAND
           61485  TCR
         4717901  T
         8430791  CELL
         2553258  RECEPTOR?
           67997  T(W)CELL(W)RECEPTOR?
          183412  REARRANGEMENT?
      S3    1207  (CD40L OR CD40 OR CD40(W)LIGAND) AND (TCR OR
                  T(W)CELL(W)RECEPTOR? OR REARRANGEMENT?)
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rearrangement?)
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            5037  CD40L
           20273  CD40
           20273  CD40
          374036  LIGAND
            9336  CD40(W)LIGAND
           61485  TCR
         4717901  T
         8430791  CELL
         2553258  RECEPTOR?
           67997  T(W)CELL(W)RECEPTOR?
          183412  REARRANGEMENT?
      S4     413  (CD40L OR CD40 OR CD40(W)LIGAND) (20N) (TCR OR
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...examined 50 records  (100)
...examined 50 records  (150)
...examined 50 records  (200)
...examined 50 records  (250)
...examined 50 records  (300)
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...examined 50 records   (350)
...examined 50 records   (400)
...completed examining records
      S5      219  RD S4 (unique items)
? s s5 and induc?(10n)(tcr or t(W)cell(W)receptor? or rearrangement)
Processing
Processing
             219  S5
         4711393  INDUC?
           61485  TCR
         4717901  T
         8430791  CELL
         2553258  RECEPTOR?
           67997  T(W)CELL(W)RECEPTOR?
          150184  REARRANGEMENT
           14578  INDUC?(10N)((TCR OR T(W)CELL(W)RECEPTOR?) OR
                  REARRANGEMENT)
      S6       47  S5 AND INDUC?(10N)(TCR OR T(W)CELL(W)RECEPTOR? OR
                  REARRANGEMENT)
? rd s6
...completed examining records
      S7       47  RD S6 (unique items)
? t s7/3/all

 7/3/1     (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
(c) 2004 BIOSIS. All rts. reserv.

0014604081   BIOSIS NO.: 200300562800
Flexible migration program regulates gammadelta T-cell involvement in
  humoral immunity.
AUTHOR: Brandes Marlene; Willimann Katharina; Lang Alois B; Nam Ki-Hoan;
  Jin Chenggang; Brenner Michael B; Morita Craig T; Moser Bernhard
  (Reprint)
AUTHOR ADDRESS: Theodor-Kocher Institute, University of Bern, CH-3000, PO
  Box 99, Bern 9, Switzerland**Switzerland
AUTHOR E-MAIL ADDRESS: bernhard.moser@tki.unibe.ch
JOURNAL: Blood  102 (10): p3693-3701 November 15, 2003 2003
MEDIUM: print
ISSN: 0006-4971
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English


 7/3/2     (Item 2 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
(c) 2004 BIOSIS. All rts. reserv.

0014557433   BIOSIS NO.: 200300512796
THE ANTIGEN PRESENTING ACTIVITY OF FRESH AND CULTURED CD45+ CELLS FROM THE
  RETINA
AUTHOR: Gregerson D S (Reprint); Sam T N (Reprint); Yang J (Reprint)
AUTHOR ADDRESS: Department of Ophthalmology, University of Minnesota,
  Minneapolis, MN, USA**USA
JOURNAL: ARVO Annual Meeting Abstract Search and Program Planner  2003 p
Abstract No. 1055 2003 2003
MEDIUM: cd-rom
CONFERENCE/MEETING: Annual Meeting of the Association for Research in
Vision and Ophthalmology  Fort Lauderdale, FL, USA  May 04-08, 2003;
20030504
SPONSOR: Association for Research in Vision and Ophthalmology
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
```

LANGUAGE: English

7/3/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0014172229 BIOSIS NO.: 200300130948
TRANCE together with IL-7 induces pre-B cells to proliferate.
AUTHOR: Kato Ibuki; Sato Hiromu; Kudo Akira (Reprint)
AUTHOR ADDRESS: Department of Life Science, Tokyo Institute of Technology, 4259 Nagatsuta, Midori-ku, Yokohama, 226-8501, Japan**Japan
AUTHOR E-MAIL ADDRESS: akudo@bio.titech.ac.jp
JOURNAL: European Journal of Immunology 33 (2): p334-341 February 2003 2003
MEDIUM: print
ISSN: 0014-2980 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

7/3/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013983901 BIOSIS NO.: 200200577412
Combined effects of calcineurin inhibitors or sirolimus with anti-CD40L mAb on alloengraftment under nonmyeloablative conditions
AUTHOR: Taylor Patricia A; Lees Christopher J; Wilson Jessica M; Ehrhardt Michael J; Campbell Matthew T; Noelle Randolph J; Blazar Bruce R (Reprint)
AUTHOR ADDRESS: University of Minnesota, 420 Delaware St SE, MMC 109, Minneapolis, MN, 55455, USA**USA
JOURNAL: Blood 100 (9): p3400-3407 November 1, 2002 2002
MEDIUM: print
ISSN: 0006-4971
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

7/3/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013763117 BIOSIS NO.: 200200356628
Analysis of maturation states of rat bone marrow-derived dendritic cells using an improved culture technique
AUTHOR: Grauer Oliver (Reprint); Wohlleben Gisela; Seubert Silvia; Weishaupt Andreas; Kaempgen Eckhart; Gold Ralf
AUTHOR ADDRESS: Department of Neurology, University of Regensburg, Universitaetsstrasse 84, 93053, Regensburg, Germany**Germany
JOURNAL: Histochemistry and Cell Biology 117 (4): p351-362 April, 2002 2002
MEDIUM: print
ISSN: 0948-6143
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

7/3/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013757933 BIOSIS NO.: 200200351444
CD40 ligation in the presence of self-reactive CD8 T cells leads to severe immunopathology
AUTHOR: Roth Evelyn; Schwartzkopff Johannes; Pircher Hanspeter (Reprint)
AUTHOR ADDRESS: Department of Immunology, Institute for Medical Microbiology and Hygiene, University of Freiburg, Hermann-Herder-Strasse 11, D-79104, Freiburg, Germany**Germany
JOURNAL: Journal of Immunology 168 (10): p5124-5129 May 15, 2002 2002
MEDIUM: print
ISSN: 0022-1767
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

7/3/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013757911 BIOSIS NO.: 200200351422
Sustained NFAT signaling promotes a Th1-like pattern of gene expression in primary murine CD4+ T cells
AUTHOR: Porter Cynthia M; Clipstone Neil A (Reprint)
AUTHOR ADDRESS: Department of Microbiology-Immunology, Medical School, Northwestern University, 303 East Chicago Avenue, Room Tarry 6-701, Chicago, IL, 60611, USA**USA
JOURNAL: Journal of Immunology 168 (10): p4936-4945 May 15, 2002 2002
MEDIUM: print
ISSN: 0022-1767
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

7/3/8 (Item 8 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013710480 BIOSIS NO.: 200200303991
Temporal sequence and functional implications of Vbeta-specific T cell receptor down-regulation and costimulatory molecule expression following in vitro stimulation with the staphylococcal superantigen toxic shock syndrome toxin-1
AUTHOR: Kum Winnie W S; Hung Ryan W Y; Cameron Scott B; Chow Anthony W (Reprint)
AUTHOR ADDRESS: Division of Infectious Diseases, G. F. Strong Research Laboratories, Vancouver Hospital and Health Sciences Center, 2733 Heather St., Vancouver, BC, V5Z3J5, Canada**Canada
JOURNAL: Journal of Infectious Diseases 185 (4): p555-560 15 February, 2002 2002
MEDIUM: print
ISSN: 0022-1899
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

7/3/9 (Item 9 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013656598 BIOSIS NO.: 200200250109

Cyclosporine A and rapamycin are synergistic for the promotion of allogeneic engraftment by anti-CD40L mAb under nonmyeloablative conditions
AUTHOR: Taylor Patricia A (Reprint); Lees Christopher J (Reprint); Noelle Randolph J; Blazar Bruce R (Reprint)
AUTHOR ADDRESS: Univ. of Minnesota Cancer Center, Univ. of Minn., Minneapolis, MN, USA**USA
JOURNAL: Blood 98 (11 Part 1): p735a-736a November 16, 2001 2001
MEDIUM: print
CONFERENCE/MEETING: 43rd Annual Meeting of the American Society of Hematology, Part 1 Orlando, Florida, USA December 07-11, 2001; 20011207
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

7/3/10 (Item 10 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013333273 BIOSIS NO.: 200100505112
N-substituted benzamides inhibit nuclear factor-kappaB and nuclear factor of activated T cells activity while inducing activator protein 1 activity in T lymphocytes
AUTHOR: Lindgren Hanna (Reprint); Pero Ronald W; Ivars Fredrik; Leanderson Tomas
AUTHOR ADDRESS: Department of Cell and Molecular Biology, Section for Immunology, Lund University, S-221 84, Lund, Sweden**Sweden
JOURNAL: Molecular Immunology 38 (4): p267-277 August, 2001 2001
MEDIUM: print
ISSN: 0161-5890
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

7/3/11 (Item 11 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013106714 BIOSIS NO.: 200100278553
Role of CD40 ligand signaling in defective type-1 cytokine response in HIV infection
AUTHOR: Subauste Carlos S (Reprint); Wessendarp Matthew (Reprint); Smulian George (Reprint); Frame Peter T (Reprint)
AUTHOR ADDRESS: University of Cincinnati College of Medicine, 231 Bethesda Av, ML0560, Cincinnati, OH, 45267-0560, USA**USA
JOURNAL: FASEB Journal 15 (4): pA308 March 7, 2001 2001
MEDIUM: print
CONFERENCE/MEETING: Annual Meeting of the Federation of American Societies for Experimental Biology on Experimental Biology 2001 Orlando, Florida, USA March 31-April 04, 2001; 20010331
ISSN: 0892-6638
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

7/3/12 (Item 12 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0012029852 BIOSIS NO.: 199900289512
Functional and phenotypic analysis of thymic B cells: Role in the induction of T cell negative selection
AUTHOR: Ferrero Isabel; Anjuere Fabienne; Martin Pilar; del Hoyo Gloria Martinez; Lopez Fraga Marta; Wright Natalia; Varona Rosa; Marquez Gabriel ; Ardavin Carlos (Reprint)
AUTHOR ADDRESS: Department of Cell Biology, Faculty of Biology, Complutense University, E-28040, Madrid, Spain**Spain
JOURNAL: European Journal of Immunology 29 (5): p1598-1609 May, 1999 1999
MEDIUM: print
ISSN: 0014-2980
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

7/3/13 (Item 13 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0011960204 BIOSIS NO.: 199900219864
Regulation of CD40L expression by cyclic AMP: Contrasting proinflammatory and inhibitory actions
AUTHOR: Wingett Denise G; Forcier Kristin; Nielson Christopher P (Reprint)
AUTHOR ADDRESS: Boise VA Medical Center, 500 W. Fort Street, Research Service 151, Boise, ID, 83702, USA**USA
JOURNAL: Cellular Immunology 192 (2): p203-212 March 15, 1999 1999
MEDIUM: print
ISSN: 0008-8749
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

7/3/14 (Item 14 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0011553658 BIOSIS NO.: 199800347905
Th1 cells induce and Th2 inhibit antigen-dependent IL-12 secretion by dendritic cells
AUTHOR: Ria Francesco; Penna Giuseppe; Adorini Luciano (Reprint)
AUTHOR ADDRESS: Roche Milano Ricerche, Via Olgettina 58, I-20132 Milano, Italy**Italy
JOURNAL: European Journal of Immunology 28 (6): p2003-2016 June, 1998 1998
MEDIUM: print
ISSN: 0014-2980
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

7/3/15 (Item 15 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0011525330 BIOSIS NO.: 199800319577
A humanised therapeutic CD4 mAb inhibits **TCR-induced** IL-2, IL-4, and IL-10 secretion and expression of CD25, **CD40L**, and CD69
AUTHOR: Woods Margaret (Reprint); Guy Robert (Reprint); Waldmann Herman; Glennie Martin; Alexander Denis R (Reprint)
AUTHOR ADDRESS: T Cell Lab., Dep. Immunol., Babraham Inst. Cambridge CB2 4AT, UK**UK
JOURNAL: Cellular Immunology 185 (2): p101-113 May 1, 1998 1998

MEDIUM: print
ISSN: 0008-8749
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

7/3/16 (Item 16 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0010464429 BIOSIS NO.: 199699098489
Functional **CD40 ligand** expression on T lymphocytes in the absence of **T cell receptor** engagement: Involvement in interleukin-2-**induced** interleukin-12 and interferon-gamma production
AUTHOR: Armant Myriam; Armitage Richard; Boiani Normal; Delespesse Guy; Sarfati Marika (Reprint)
AUTHOR ADDRESS: Allergy Res. Lab., Louis-Charles Simard Res. Cent., Notre-Dame-Hospital, 1560 Sherbrooke Street East, Montreal, PQ H2L 4M1, Canada**Canada
JOURNAL: European Journal of Immunology 26 (7): p1430-1434 1996 1996
ISSN: 0014-2980
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

7/3/17 (Item 17 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0010276433 BIOSIS NO.: 199698744266
Regulation of **CD40 ligand** expression on naive CD4 T cells: A role for **TCR** but not co-stimulatory signals
AUTHOR: Jaiswal Archana I; Dubey Caroline; Swain Susan L; Croft Michael (Reprint)
AUTHOR ADDRESS: Cancer Center, Univ. California San Diego, 9500 Gilman Dr., La Jolla, CA 92093-0063, USA**USA
JOURNAL: International Immunology 8 (2): p275-285 1996 1996
ISSN: 0953-8178
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

7/3/18 (Item 18 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0010260383 BIOSIS NO.: 199698728216
Parietaria judaica-specific T-cell clones from atopic patients: Heterogeneity in restriction, V-beta usage, and cytokine profile
AUTHOR: Sallusto Federica; Corinti Silvia; Pini Carlo; Biocca Marina M; Bruno Guglielmo; Felice Gabriella Di (Reprint)
AUTHOR ADDRESS: Dep. Immunol., Ist. Superior Sanita, V.le Regina Elena, 299, 00161 Rome, Italy**Italy
JOURNAL: Journal of Allergy and Clinical Immunology 97 (2): p627-637 1996 1996
ISSN: 0091-6749
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

7/3/19 (Item 19 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0010095661 BIOSIS NO.: 199698563494
Altered CD40 ligand induction in tolerant T lymphocytes
AUTHOR: Bowen Frank; Haluskey Joyce; Quill Helen (Reprint)
AUTHOR ADDRESS: DAIT, NIAID, NIH, 6003 Executive Blvd., Bethesda, MD 20892, USA**USA
JOURNAL: European Journal of Immunology 25 (10): p2830-2834 1995 1995
ISSN: 0014-2980
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

7/3/20 (Item 20 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0009203005 BIOSIS NO.: 199497224290
CD40 ligand acts as a costimulatory signal for neonatal thymic gamma-delta T cells
AUTHOR: Ramsdell Fred (Reprint); Seaman Michael S; Clifford K N; Fanslow William C
AUTHOR ADDRESS: Dep. Immunobiol., Immunex Res. Dev. Corp., 51 University St., Seattle, WA 98101, USA**USA
JOURNAL: Journal of Immunology 152 (5): p2190-2197 1994 1994
ISSN: 0022-1767
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

7/3/21 (Item 21 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0009053879 BIOSIS NO.: 199497075164
Ligation of B7 with CD28/CTLA-4 on T cells results in CD40 ligand expression, interleukin-4 secretion and efficient help for antibody production by B cells
AUTHOR: De Boer Mark (Reprint); Kasran Ahmad; Kwekkeboom Jaap; Walter Hugo; Vandenberghe Peter; Ceuppens Jan L
AUTHOR ADDRESS: Dep. Immunol., Innogenetics N.V., Industriepark Zwijnaarde 7, Box 4, B-9052 Ghent, Belgium**Belgium
JOURNAL: European Journal of Immunology 23 (12): p3120-3125 1993 1993
ISSN: 0014-2980
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

7/3/22 (Item 22 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0008210564 BIOSIS NO.: 199293053455
TUMOR NECROSIS FACTOR TNF RECEPTOR EXPRESSION IN T LYMPHOCYTES DIFFERENTIAL REGULATION OF THE TYPE I TNF RECEPTOR DURING ACTIVATION OF RESTING AND EFFECTOR T CELLS
AUTHOR: WARE C F (Reprint); CROWE P D; VANARSDALE T L; ANDREWS J L; GRAYSON M H; JERZY R; SMITH C A; GOODWIN R G

AUTHOR ADDRESS: DIV BIOMED SCI, UNIV CALIFORNIA, RIVERSIDE, CALIF 92521, USA**USA
JOURNAL: Journal of Immunology 147 (12): p4229-4238 1991
ISSN: 0022-1767
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: ENGLISH

7/3/23 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

12019226 EMBASE No: 2003129970
Cutting edge: CD40-induced expression of recombination activating gene (RAG) 1 and RAG2: A mechanism for the generation of autoaggressive T cells in the periphery
Vaitaitis G.M.; Poulin M.; Sanderson R.J.; Haskins K.; Wagner Jr. D.H.
Dr. D.H. Wagner Jr., Webb-Waring Inst. Cancer/Aging Res., Health Sciences Center, University of Colorado, 4200 East Ninth Avenue, Denver, CO 80262 United States
AUTHOR EMAIL: david.wagner@uchsc.edu
Journal of Immunology (J. IMMUNOL.) (United States) 01 APR 2003, 170/7 (3455-3459)
CODEN: JOIMA ISSN: 0022-1767
DOCUMENT TYPE: Journal ; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 24

7/3/24 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE

11606223 EMBASE No: 2002170539
Sustained NFAT signaling promotes a Th1-like pattern of gene expression in primary murine CD4SUP+ T cells
Porter C.M.; Clipstone N.A.
Dr. N.A. Clipstone, Department of Microbiology, Northwestern Univ. Medical School, 303 East Chicago Avenue, Chicago, IL 60611 United States

AUTHOR EMAIL: n-clipstone@northwestern.edu
Journal of Immunology (J. IMMUNOL.) (United States) 15 MAY 2002, 168/10 (4936-4945)
CODEN: JOIMA ISSN: 0022-1767
DOCUMENT TYPE: Journal ; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 64

7/3/25 (Item 3 from file: 73)
DIALOG(R)File 73:EMBASE

11101671 EMBASE No: 2001112786
Regulation of transcriptional activity of the murine **CD40 ligand** promoter in response to signals through **TCR** and the costimulatory molecules CD28 and CD2
Lindgren H.; Axcrona K.; Leanderson T.
H. Lindgren, Section for Immunology, Dept. of Cell and Molecular Biology, Lund University, Solvegatan 21, S-223 62 Lund Sweden
AUTHOR EMAIL: hanna.lindgren@immuno.lu.se
Journal of Immunology (J. IMMUNOL.) (United States) 01 APR 2001, 166/7 (4578-4585)

CODEN: JOIMA ISSN: 0022-1767
DOCUMENT TYPE: Journal ; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 56

7/3/26 (Item 4 from file: 73)
DIALOG(R)File 73:EMBASE

11062183 EMBASE No: 2001078874
Identification of a CD28 response element in the CD40 ligand promoter
Parra E.; Mustelin T.; Dohlsten M.; Mercola D.
Dr. E. Parra, Sidney Kimmel Cancer Center, 10835 Altman Row, San Diego, CA 92121 United States
AUTHOR EMAIL: eparra@skcc.org
Journal of Immunology (J. IMMUNOL.) (United States) 15 FEB 2001, 166/4 (2437-2443)
CODEN: JOIMA ISSN: 0022-1767
DOCUMENT TYPE: Journal ; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 49

7/3/27 (Item 5 from file: 73)
DIALOG(R)File 73:EMBASE

11035624 EMBASE No: 2001048816
Broad programming by IL-2 receptor signaling for extended growth to multiple cytokines and functional maturation of antigen-activated T cells
Malek T.R.; Yu A.; Scibelli P.; Lichtenheld M.G.; Codias E.K.
Dr. T.R. Malek, Dept. of Microbiology and Immunology, Univ. of Miami School of Medicine, P.O. Box 016960 (R138), Miami, FL 33101 United States
AUTHOR EMAIL: tmalek@med.miami.edu
Journal of Immunology (J. IMMUNOL.) (United States) 01 FEB 2001, 166/3 (1675-1683)
CODEN: JOIMA ISSN: 0022-1767
DOCUMENT TYPE: Journal ; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 46

7/3/28 (Item 6 from file: 73)
DIALOG(R)File 73:EMBASE

07749049 EMBASE No: 1999231072
CD40-CD154 interaction and IFN-gamma are required for IL-12 but not prostaglandin Einf 2 secretion by microglia during antigen presentation to Th1 cells
Aloisi F.; Penna G.; Polazzi E.; Minghetti L.; Adorini L.
Dr. F. Aloisi, Neurophysiology Unit, Lab. of Organ/System Pathophysiology, Istituto Superiore di Sanita, Viale Regina Elena 299, 00161 Rome Italy
Journal of Immunology (J. IMMUNOL.) (United States) 01 FEB 1999, 162/3 (1384-1391)
CODEN: JOIMA ISSN: 0022-1767
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 58

7/3/29 (Item 7 from file: 73)
DIALOG(R)File 73:EMBASE

07740269 EMBASE No: 1999217790
TCR vaccines against T cell lymphoma: QS-21 and IL-12 adjuvants induce a protective CD8sup + T cell response
Wong C.P.; Okada C.Y.; Levy R.
Dr. C.P. Wong, Department of Medicine, Division of Oncology, Stanford University Medical Center, Stanford, CA 94305 United States
AUTHOR EMAIL: pscarmen@leland.stanford.edu
Journal of Immunology (J. IMMUNOL.) (United States) 15 FEB 1999, 162/4 (2251-2258)
CODEN: JOIMA ISSN: 0022-1767
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 50

7/3/30 (Item 8 from file: 73)
DIALOG(R)File 73:EMBASE

07411640 EMBASE No: 1998313042
Signaling through a CD3gamma-deficient TCR/CD3 complex in immortalized mature CD4sup + and CD8sup + T lymphocytes
Pacheco-Castro A.; Alvarez-Zapata D.; Serrano-Torres P.; Regueiro J.R.
Dr. J.R. Regueiro, Facultad de Medicina, Universidad Complutense, 28040 Madrid Spain
AUTHOR EMAIL: regueiro@eucmax.sim.ucm.es
Journal of Immunology (J. IMMUNOL.) (United States) 15 SEP 1998, 161/6 (3152-3160)
CODEN: JOIMA ISSN: 0022-1767
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 48

7/3/31 (Item 9 from file: 73)
DIALOG(R)File 73:EMBASE

07339520 EMBASE No: 1998235639
Anti-CD40L accelerates renal disease and adenopathy in MRL-lpr mice in parallel with decreased thymocyte apoptosis
Russell J.Q.; Mooney T.; Cohen P.L.; MacPherson B.; Noelle R.J.; Budd R.C.
Dr. R.C. Budd, Univ. of Vermont College of Medicine, Given Medical Building C-303, Burlington, VT 05405 United States
AUTHOR EMAIL: rbudd@pop.uvm.edu
Journal of Immunology (J. IMMUNOL.) (United States) 15 JUL 1998, 161/2 (729-739)
CODEN: JOIMA ISSN: 0022-1767
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 49

7/3/32 (Item 10 from file: 73)
DIALOG(R)File 73:EMBASE

06403974 EMBASE No: 1996067029
Parietaria judaica-specific T-cell clones from atopic patients:

Heterogeneity in restriction, Vbeta usage, and cytokine profile
Sallusto F.; Corinti S.; Pini C.; Biocca M.M.; Bruno G.; Di Felice G.
Department of Immunology, Istituto Superiore di Sanita, V.le Regina Elena, 299,00161-Rome Italy
Journal of Allergy and Clinical Immunology (J. ALLERGY CLIN. IMMUNOL.) (United States) 1996, 97/2 (627-637)
CODEN: JACIB ISSN: 0091-6749
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

7/3/33 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

14484096 PMID: 10481375
[Regulation of the contact sensitivity reaction by suppression of T gamma delta lymphocytes]
Regulacja reakcji nadwrazliwosci kontaktowej przez supresyjne limfocyty T gamma delta.
Szczepanik M
Katedra Immunologii Collegium Medicum UJ w Krakowie.
Folia medica Cracoviensia (POLAND) 1998, 39 (1-2) p5-33, ISSN 0015-5616 Journal Code: 0374617
Document type: Journal Article; Review; Review, Tutorial ; English Abstract
Languages: POLISH
Main Citation Owner: NLM
Record type: Completed

7/3/34 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

14430614 PMID: 10425271
Increased expression of **CD40** on thymocytes and peripheral T cells in autoimmunity: a mechanism for acquiring changes in the peripheral ***T*** ***cell*** ***receptor*** repertoire.
Wagner D H; Newell E; Sanderson R J; Freed J H; Newell M K
Webb-Waring Institute for Cancer, Aging and Antioxidant Research, Denver, CO 80262, USA.
International journal of molecular medicine (GREECE) Sep 1999, 4 (3) p231-42, ISSN 1107-3756 Journal Code: 9810955
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

7/3/35 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

14263946 PMID: 10083602
CD40 upregulation in **TCR** alpha/beta+ CD68+ cells and parenchymal **CD40L induction** and associated with NF-kappa B activation in chronic rejecting human renal allografts.
Gaweco A S; Mitchell B L; Lucas B; McClatchey K D; Van Thiel D H
Department of Medicine, Loyola University Medical Center, Loyola University Chicago, Maywood, IL 60153, USA. agaweco@luc.edu
Transplantation proceedings (UNITED STATES) Feb-Mar 1999, 31 (1-2) p1359-60, ISSN 0041-1345 Journal Code: 0243532
Document type: Journal Article

Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

7/3/36 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

14221068 PMID: 9973501
TCR vaccines against T cell lymphoma: QS-21 and IL-12 adjuvants induce a protective CD8+ T cell response.
Wong C P; Okada C Y; Levy R
Department of Medicine, Division of Oncology, Stanford University School of Medicine, CA 94305, USA. pscarmen@leland.stanford.edu
Journal of immunology (Baltimore, Md. - 1950) (UNITED STATES) Feb 15 1999, 162 (4) p2251-8, ISSN 0022-1767 Journal Code: 2985117R
Contract/Grant No.: CA 69521; CA; NCI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

7/3/37 (Item 5 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

14220960 PMID: 9973393
CD40-CD154 interaction and IFN-gamma are required for IL-12 but not prostaglandin E2 secretion by microglia during antigen presentation to Th1 cells.
Aloisi F; Penna G; Polazzi E; Minghetti L; Adorini L
Laboratory of Organ and System Pathophysiology, Istituto Superiore di Sanita, Rome, Italy.
Journal of immunology (Baltimore, Md. - 1950) (UNITED STATES) Feb 1 1999, 162 (3) p1384-91, ISSN 0022-1767 Journal Code: 2985117R
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

7/3/38 (Item 6 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

14042592 PMID: 9743383
Signaling through a CD3 gamma-deficient TCR/CD3 complex in immortalized mature CD4+ and CD8+ T lymphocytes.
Pacheco-Castro A; Alvarez-Zapata D; Serrano-Torres P; Regueiro J R
Immunologia, Facultad de Medicina, Universidad Complutense, Madrid, Spain.
Journal of immunology (Baltimore, Md. - 1950) (UNITED STATES) Sep 15 1998, 161 (6) p3152-60, ISSN 0022-1767 Journal Code: 2985117R
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

7/3/39 (Item 7 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13028674 PMID: 8724831
Regulation of T lymphocyte subsets.
Fitch F W; Stack R; Fields P; Lancki D W; Cronin D C
Department of Pathology, University of Chicago, IL 60637, USA.
Ciba Foundation symposium (NETHERLANDS) 1995, 195 p68-80; discussion 80-5, ISSN 0300-5208 Journal Code: 0356636
Contract/Grant No.: AI-29531; AI; NIAID; AI-35294; AI; NIAID; CA-44372; CA; NCI
Document type: Journal Article; Review; Review, Tutorial
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

7/3/40 (Item 8 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

12630788 PMID: 7751632
Regulation of early T cell development by the engagement of TCR-beta complex expressed on fetal thymocytes from TCR-beta-transgenic scid mice.
Takahama Y; Sugaya K; Tsuda S; Hasegawa T; Hashimoto Y
Institute of Immunology, Syntex, Ibaraki, Japan.
Journal of immunology (Baltimore, Md. - 1950) (UNITED STATES) Jun 1 1995, 154 (11) p5862-9, ISSN 0022-1767 Journal Code: 2985117R
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

7/3/41 (Item 9 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

11690780 PMID: 11865410
Temporal sequence and functional implications of V beta-specific T cell receptor down-regulation and costimulatory molecule expression following in vitro stimulation with the staphylococcal superantigen Toxic shock syndrome toxin-1.
Kum Winnie W S; Hung Ryan W Y; Cameron Scott B; Chow Anthony W
Department of Medicine, Division of Infectious Diseases, University of British Columbia, Vancouver Hospital, Vancouver, Canada.
Journal of infectious diseases (United States) Feb 15 2002, 185 (4) p555-60, ISSN 0022-1899 Journal Code: 0413675
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

7/3/42 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

136261819 CA: 136(17)261819j PATENT
Coupling of peripheral tolerance to endogenous IL-10 promotes effective modulation of T cells and ameliorates autoimmune disease
INVENTOR(AUTHOR): Zaghouani, Habib
LOCATION: USA
PATENT: U.S. Pat. Appl. Publ. ; US 20020038002 A1 DATE: 20020328
APPLICATION: US 873901 (20010604) *US PV209527 (20000605)
PAGES: 84 pp. CODEN: USXXCO LANGUAGE: English CLASS: 530388220;

A61K-039/395A; C07K-016/28B

7/3/43 (Item 2 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

132077527 CA: 132(7)77527d JOURNAL
Essential role for both CD80 and CD86 costimulation, but not CD40 interactions, in allergen-induced Th2 cytokine production from asthmatic bronchial tissue: role for αβ, but not γδ, T cells
AUTHOR(S): Jaffar, Zeina H.; Stanciu, Luminita; Pandit, Anita; Lordan, James; Holgate, Stephen T.; Roberts, Kevan
LOCATION: Southampton General Hospital, University Medicine, Southampton, UK, SO16 6YD
JOURNAL: J. Immunol. DATE: 1999 VOLUME: 163 NUMBER: 11 PAGES: 6283-6291 CODEN: JOIMA3 ISSN: 0022-1767 LANGUAGE: English PUBLISHER: American Association of Immunologists

7/3/44 (Item 3 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

131086583 CA: 131(7)86583a JOURNAL
A Role for CD99 in T Cell Activation
AUTHOR(S): Wingett, Denise; Forcier, Kristin; Nielson, Christopher P.
LOCATION: Department of Veterans Affairs Medical Center, Boise, ID, 83702, USA
JOURNAL: Cell. Immunol. DATE: 1999 VOLUME: 193 NUMBER: 1 PAGES: 17-23
CODEN: CLIMB8 ISSN: 0008-8749 LANGUAGE: English PUBLISHER: Academic Press

7/3/45 (Item 4 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

129134960 CA: 129(11)134960d JOURNAL
A humanized therapeutic CD4 mAb inhibits TCR-induced IL-2, IL-4, and IL-10 secretion and expression of CD25, CD40L, and CD69
AUTHOR(S): Woods, Margaret; Guy, Robert; Waldmann, Herman; Glennie, Martin; Alexander, Denis R.
LOCATION: T Cell Laboratory, Department of Immunology, Babraham Inst., Cambridge, UK, CB2 4AT
JOURNAL: Cell. Immunol. DATE: 1998 VOLUME: 185 NUMBER: 2 PAGES: 101-113 CODEN: CLIMB8 ISSN: 0008-8749 LANGUAGE: English PUBLISHER: Academic Press

7/3/46 (Item 5 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

128216369 CA: 128(18)216369m PATENT
Bi- and trispecific antibodies for induction of tumor immunity
INVENTOR(AUTHOR): Lindhofer, Horst; Kolb, Hans-Jochem; Thierfelder, Stefan
LOCATION: Germany,
ASSIGNEE: GSF-Forschungszentrum fuer Umwelt und Gesundheit G.m.b.H. Neuherberg
PATENT: Germany Offen. ; DE 19710497 A1 DATE: 19980305
APPLICATION: DE 19710497 (19970313) *DE 19635743 (19960903) *DE 19648976 (19961126)

PAGES: 18 pp. CODEN: GWXXBX LANGUAGE: German CLASS: C07K-016/28A; A61K-039/395B

7/3/47 (Item 6 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

122237721 CA: 122(19)237721m JOURNAL
A subset of CD4+ memory T cells contains preformed CD40 ligand that is rapidly but transiently expressed on their surface after activation through the T cell receptor complex
AUTHOR(S): Palleja, Casamayor, Montserrat; Khan, Mahmood; MacLennan, Ian C. M.
LOCATION: Dep. Immunology, Univ. Birmingham Med. Sch., Birmingham, UK, B15 2TT
JOURNAL: J. Exp. Med. DATE: 1995 VOLUME: 181 NUMBER: 4 PAGES: 1293-301 CODEN: JEMEAV ISSN: 0022-1007 LANGUAGE: English
? t s7/7/all

7/7/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0014604081 BIOSIS NO.: 200300562800
Flexible migration program regulates gammadelta T-cell involvement in humoral immunity.
AUTHOR: Brandes Marlene; Willimann Katharina; Lang Alois B; Nam Ki-Hoan; Jin Chenggang; Brenner Michael B; Morita Craig T; Moser Bernhard (Reprint)
AUTHOR ADDRESS: Theodor-Kocher Institute, University of Bern, CH-3000, PO Box 99, Bern 9, Switzerland**Switzerland
AUTHOR E-MAIL ADDRESS: bernhard.moser@tki.unibe.ch
JOURNAL: Blood 102 (10): p3693-3701 November 15, 2003 2003
MEDIUM: print
ISSN: 0006-4971
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: gammadelta T cells are inadequately defined both in terms of their migration potential and contribution to antimicrobial immunity. Here, we have examined the migration profile of human blood gammadelta T cells and related cell lines and correlated these findings with their distribution in secondary lymphoid tissues and their function in B-cell cocultures. We find that resting gammadelta T cells are characterized by an inflammatory migration program similar to cells of the innate immune system. However, ***T*** - ***cell*** ***receptor*** (***TCR***) triggering resulted in the rapid but transient **induction** of a lymph node (LN)-homing program, as evidenced by functional CCR7 expression and concomitant reduction in expression and function of CCR5 and, to a lesser degree, CCR2. Moreover, the LN-homing program was reflected by the presence of gammadelta T cells in gastrointestinal lymphoid tissues, notably in clusters within germinal centers of B-cell follicles. In line with these findings, VgammaVdelta-**TCR** triggering resulted in prominent expression of essential B-cell costimulatory molecules, including ***CD40L*** , OX40, CD70, and ICOS. Furthermore, gammadelta T cells were shown to provide potent B-cell help during in vitro antibody production. Collectively, our findings agree with a role for gammadelta T cells in humoral immunity during the early phase of antimicrobial responses.

7/7/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0014557433 BIOSIS NO.: 200300512796
THE ANTIGEN PRESENTING ACTIVITY OF FRESH AND CULTURED CD45+ CELLS FROM THE RETINA
AUTHOR: Gregerson D S (Reprint); Sam T N (Reprint); Yang J (Reprint)
AUTHOR ADDRESS: Department of Ophthalmology, University of Minnesota, Minneapolis, MN, USA**USA
JOURNAL: ARVO Annual Meeting Abstract Search and Program Planner 2003 p Abstract No. 1055 2003 2003
MEDIUM: cd-rom
CONFERENCE/MEETING: Annual Meeting of the Association for Research in Vision and Ophthalmology Fort Lauderdale, FL, USA May 04-08, 2003; 20030504
SPONSOR: Association for Research in Vision and Ophthalmology
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Purpose: Several lines of investigation indicate that Ag recognition takes place in the retina, or that retina-derived Ag is recognized elsewhere. To explore the possibility that cells from the retina are capable of presenting Ag directly to CD4 T cells, CD45+ cells were isolated from retina and tested in vitro for the ability to present specific Ag to naive and Ag-experienced CD4 T cells. Methods: Retinas were harvested and enzymatically dissociated. The cell suspension was separated on a density gradient, and the buoyant cells positively selected with anti-CD45 by magnetic cell separation. The resulting population was cultured with naive and Ag-experienced **TCR**-Tg CD4 T cells specific for beta-galactosidase. In some cases, the retinal cells were activated with IFN-gamma, anti- ***CD40*** or LPS. Spleen cells, or fractions of spleen cells were used as positive controls for Ag presentation. The T cells were recovered and analyzed by flow cytometry to determine their numbers and activation status. Measures of activation including changes in CD44, CD45RB, CD62L, CD69 and size. In some experiments, T cells were labeled with CFSE prior to culture. Results: Retinal CD45+ cells were poorly able to support an Ag-dependent proliferative response in both naive and antigen-experienced T cells, unlike the spleen cells, which supported a potent response in both populations. Activation of the retinal cells in vitro with IFN-gamma, anti-CD40 or LPS increased their activity as APC to a small degree, but it remained well below the activation observed in parallel cultures with spleen cells or CD11c-enriched splenic APC. Addition of retinal CD45+ cells to co-cultures of T cells and spleen cells reduced the proliferative response of the T cells. Analysis of activation markers on the T cells cultured with Ag and the retinal CD45+ cells revealed small differences, suggesting that some ligation of the TCR had taken place, but that little proliferation resulted from that interaction. Conclusions: Although some CD45+ cells from the retina express class II MHC, and costimulatory molecules (CD80), the ability of these cells to support Ag-dependent proliferative responses in Ag-specific T cells is minimal. Their APC activity is not much enhanced by pre-treatment with activators including IFN-gamma, anti- ***CD40*** and LPS. Since they give some evidence of the ability to ligate the **TCR**, we speculate that they may be ***inducing*** a regulatory response in the T cells.

7/7/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0014172229 BIOSIS NO.: 200300130948

TRANCE together with IL-7 induces pre-B cells to proliferate.
AUTHOR: Kato Ibuki; Sato Hiromu; Kudo Akira (Reprint)
AUTHOR ADDRESS: Department of Life Science, Tokyo Institute of Technology, 4259 Nagatsuta, Midori-ku, Yokohama, 226-8501, Japan**Japan
AUTHOR E-MAIL ADDRESS: akudo@bio.titech.ac.jp
JOURNAL: European Journal of Immunology 33 (2): p334-341 February 2003 2003
MEDIUM: print
ISSN: 0014-2980 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: TRANCE (TNF-related activation-induced cytokine)-deficient mice completely lack osteoclasts, and develop severe osteopetrosis. These mice also show a defect in their pre-B cell differentiation. In the present study, the expression of TRANCE was examined in pre-B cell lines using flow cytometry and reverse transcription-PCR. Three pre-B cell lines, 18-81, B3P816-1, and 38B9, expressed TRANCE on their surface, and two pre-B cell lines, 70Z/3 and NFS5, at the late pre-B cell stage, expressed it at low levels, although their mRNA expression was normal. Another pre-B cell line, 38-C-13, at the intermediate stage between pre-B and immature B cells, did not express TRANCE. The IL-7-dependent pre-B cell line PreBR, which expresses the pre-B cell receptor on the cell surface, also expressed TRANCE. When differentiation of PreBR cells was induced in vitro by removing IL-7 from cultures, TRANCE expression dropped; it was restored by the addition of IL-7, suggesting that TRANCE functions in cooperation with IL-7. To examine the function of TRANCE, we introduced the TRANCE gene into PreBR cells and established two transfectants that constitutively expressed TRANCE, even in the absence of IL-7. In these transfectants, after removal of IL-7, the number of cells that succeeded in kappa chain **rearrangement** was decreased to one third; and ***CD40*** expression decreased to less than one tenth. Moreover, the percentage of cells in the S/G2/M phase was increased by 50% over the mock transfectant. These findings indicate that, before kappa chain **rearrangement** occurs, TRANCE together with IL-7 **induces** pre-B cells to proliferate and makes this ***rearrangement*** more efficient.

7/7/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013983901 BIOSIS NO.: 200200577412
Combined effects of calcineurin inhibitors or sirolimus with anti-CD40L mAb on alloengraftment under nonmyeloablative conditions
AUTHOR: Taylor Patricia A; Lees Christopher J; Wilson Jessica M; Ehrhardt Michael J; Campbell Matthew T; Noelle Randolph J; Blazar Bruce R (Reprint)
AUTHOR ADDRESS: University of Minnesota, 420 Delaware St SE, MMC 109, Minneapolis, MN, 55455, USA**USA
JOURNAL: Blood 100 (9): p3400-3407 November 1, 2002 2002
MEDIUM: print
ISSN: 0006-4971
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The immunosuppressive drugs, cyclosporine A (CsA), tacrolimus, or sirolimus, were analyzed as single agents and in combination with anti-CD40L monoclonal antibody (mAb) for their effects on alloengraftment in mice conditioned with minimal total body irradiation (TBI). Whereas anti-CD40L mAb facilitated chimerism, neither sirolimus nor CsA resulted in substantial alloengraftment. However, sirolimus was synergistic with

anti- ***CD40L*** mAb for ***inducing*** donor chimerism. Contrary to expectations, CsA, a **T-cell receptor** (**TCR**) signaling inhibitor, did not abrogate anti-**CD40L** mAb-facilitated engraftment but rather increased engraftment in anti-**CD40L** mAb-treated mice. Although tacrolimus alone or with anti-CD40L mAb resulted in similar levels of donor chimerism, donor T-cell reconstitution was very low in tacrolimus-treated mice. At 1 week after transplantation, CsA decreased thymic numbers more profoundly than sirolimus or tacrolimus in anti-CD40L mAb-treated recipients. In contrast, only sirolimus resulted in a decrease in host splenic T-cell numbers in anti-CD40L mAb-treated recipients. Importantly, sirolimus and anti-CD40L mAb induced profound donor tolerance with 100% acceptance of donor skin grafts placed early after bone marrow transplantation (BMT). In contrast, anti-CD40L mAb alone or in combination with CsA resulted in 12% or less donor skin graft acceptance early (1 month) and 60% or less later (3 months) after BMT. These data have clinical relevance and indicate that immunosuppressive pharmacologic agents enhance anti-CD40L mAb-facilitated alloengraftment and tolerance induction under nonmyeloablative conditioning.

7/7/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013763117 BIOSIS NO.: 200200356628
Analysis of maturation states of rat bone marrow-derived dendritic cells using an improved culture technique
AUTHOR: Grauer Oliver (Reprint); Wohlleben Gisela; Seubert Silvia; Weishaupt Andreas; Kaempgen Eckhart; Gold Ralf
AUTHOR ADDRESS: Department of Neurology, University of Regensburg, Universitaetsstrasse 84, 93053, Regensburg, Germany**Germany
JOURNAL: Histochemistry and Cell Biology 117 (4): p351-362 April, 2002 2002
MEDIUM: print
ISSN: 0948-6143
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: In this study, we examined in more detail the development of rat bone marrow-derived dendritic cells (BMDC). A two-stage culture system was used to propagate BMDC from rat bone marrow precursors. BMDC developed within clusters of proliferating cells after repetitive addition of rat granulocyte/macrophage colony-stimulating factor and rat interleukin (IL)-4 at a concentration of 5 ng/ml to the cultures. Fluorescence-activated cell sorter analysis performed at an early stage of development (day 6) revealed an immature phenotype with intermediate levels of major histocompatibility complex (MHC) class II expression and low levels of the costimulator molecules CD80 and CD86. Upon further culture, a strong upregulation of MHC class II, costimulatory and adhesion molecules could be observed, whereas macrophage marker antigens were downregulated. Late-stage BMDC (day 10) showed a high expression of MHC class I and II, ICAM-1, Ox62 and CD11c, and revealed a split pattern of B7-1 and B7-2. The cell yield was about 40% of the initially plated bone marrow cells with 80% MHC class II-high and less than 20% MHC class II-low positive cells. Full maturation of rat BMDC (day 12) with an almost uniform expression of B7 was achieved by subsequent subculture and further stimulation with rat tumour necrosis factor alpha (TNF-alpha), lipopolysaccharide (LPS) or soluble CD40 ligand (CD40L). Analysis of the cell supernatant revealed a strong IL-12 production after LPS or CD40L, and to a lesser extent after TNF-alpha stimulation. Additionally, LPS-treated, but not **CD40L**-treated BMDC secreted TNF-alpha into the supernatant. Early-stage BMDC sufficiently triggered a ***T*** ***cell***

receptor (**TCR**) downregulation, but did not stimulate naive T cells in an allogeneic mixed leukocyte reaction (MLR) and revealed a low stimulatory capacity in an antigen-specific T cell assay. In contrast, late-stage BMDC and especially fully mature BMDC strongly **induced** **TCR** internalisation, elicited high T cell responses in the allogeneic MLR similar to those obtained by mature rat spleen dendritic cells and efficiently activated antigen-specific T cells. In conclusion, this protocol allows easy access to large numbers of rat BMDC at defined maturation stages and selective studies for the manipulation of immune responses in rat models.

7/7/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013757933 BIOSIS NO.: 200200351444
CD40 ligation in the presence of self-reactive CD8 T cells leads to severe immunopathology
AUTHOR: Roth Evelyn; Schwartzkopff Johannes; Pircher Hanspeter (Reprint)
AUTHOR ADDRESS: Department of Immunology, Institute for Medical Microbiology and Hygiene, University of Freiburg, Hermann-Herder-Strasse 11, D-79104, Freiburg, Germany**Germany
JOURNAL: Journal of Immunology 168 (10): p5124-5129 May 15, 2002 2002
MEDIUM: print
ISSN: 0022-1767
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Previous work has shown that stimulation of APCs via CD40 strongly influences the outcome of a CD8 T cell response. In this study, we examined the effect of CD40 ligation on peripheral tolerance induction of self-reactive CD8 T cells in an adoptive transfer model. Naive CD8 T cells from TCR-transgenic (tg) mice specific for the gp33 epitope of lymphocytic choriomeningitis virus were tolerized when transferred into H8-tg mice expressing the gp33 epitope under the control of a MHC class I promoter. However, if the H8 recipient mice were treated with agonistic anti-**CD40** Abs, **TCR**-tg cells vigorously proliferated, and ***induced*** destruction of lymphoid organs and hepatitis. Break of peripheral tolerance induction was B cell independent and did not require CD28/B7 interactions. These findings provide further in vivo evidence for the crucial role of the activation state of the APC in peripheral tolerance induction and suggest the need for caution in systemically activating APC via CD40 ligation in the presence of self-reactive T cells.

7/7/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013757911 BIOSIS NO.: 200200351422
Sustained NFAT signaling promotes a Th1-like pattern of gene expression in primary murine CD4+ T cells
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JOURNAL: Journal of Immunology 168 (10): p4936-4945 May 15, 2002 2002
MEDIUM: print
ISSN: 0022-1767
DOCUMENT TYPE: Article
RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: T cell activation is known to be critically regulated by the extent and duration of ***TCR*** - ***induced*** signaling pathways. The NFAT family of transcription factors is believed to play an important role in coupling these quantitative differences in **TCR**-***induced*** signaling events into changes in gene expression. In this study we have specifically investigated the effects of sustained NFAT signaling on T cell activation by introducing a constitutively active mutant version of NFATc1 (caNFATc1) into primary murine CD4+ T cells and examining its effects on gene expression. We now report that ectopic expression of caNFATc1 partially mimics **TCR** signaling, resulting in enhanced expression of CD25 and **CD40 ligand** and down-regulation of CD62L. More importantly, we find that expression of caNFATc1 in T cells maintained under either nonpolarizing or Th1-skewing conditions leads to a marked selective increase in the number of cells expressing the prototypical Th1 cytokine, IFN-gamma. Furthermore, when expressed in Th2-skewed cells, caNFATc1 appears to attenuate Th2 differentiation by decreasing production of IL-4 and promoting the expression of IFN-gamma. Finally, we find that caNFATc1 enhances expression of functional P-selectin glycoprotein ligand-1, up-regulates Fas ligand expression, and increases susceptibility to activation-induced cell death, cellular traits that are preferentially associated with Th1 effector cells. Taken together, these results suggest that sustained NFAT signaling, mediated by ectopic expression of caNFATc1, acts to promote a Th1-like pattern of gene expression and thereby serves to highlight the important relationship between the degree of NFAT signaling and the qualitative pattern of gene expression induced during T cell activation.

7/7/8 (Item 8 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013710480 BIOSIS NO.: 200200303991
Temporal sequence and functional implications of Vbeta-specific T cell receptor down-regulation and costimulatory molecule expression following in vitro stimulation with the staphylococcal superantigen toxic shock syndrome toxin-1
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JOURNAL: Journal of Infectious Diseases 185 (4): p555-560 15 February, 2002 2002
MEDIUM: print
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RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The superantigen toxic shock syndrome toxin-1 (TSST-1) is implicated as the major cause of staphylococcal toxic shock syndrome. The temporal sequence of early signaling events in human peripheral blood mononuclear cells following TSST-1 stimulation was examined. TSST-1 **induced** rapid and complete down-regulation of Vbeta2-specific **T cell receptor** (**TCR**), followed by transient CD154 expression on CD4+ lymphocytes. This was sequentially followed by the up-regulation of CD86, CD80, **CD40**, and human leukocyte antigen-DR expression on CD14+ monocytes. In contrast, S14N, a TSST-1 mutant toxin with a single amino acid substitution that is known to be impaired in interleukin (IL)-2, interferon (IFN)-gamma, and tumor necrosis factor (TNF)-alpha secretion, was deficient in both Vbeta2-TCR

down-regulation and CD154 and CD80/CD86 expression. Furthermore, pretreatment with monoclonal antibodies against Vbeta2-**TCR**, CD80/CD86, and CD154 significantly inhibited TSST-1-**induced** IL-2, IFN-gamma, and TNF-alpha secretion. Taken together, these results indicate that early Vbeta-specific TCR activation, along with CD80/CD86 and CD154 costimulation, are key determinants of the TSST-1-induced proinflammatory cytokine response.

7/7/9 (Item 9 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013656598 BIOSIS NO.: 200200250109
Cyclosporine A and rapamycin are synergistic for the promotion of allogeneic engraftment by anti-CD40L mAb under nonmyeloablative conditions
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AUTHOR ADDRESS: Univ. of Minnesota Cancer Center, Univ. of Minn., Minneapolis, MN, USA**USA
JOURNAL: Blood 98 (11 Part 1): p735a-736a November 16, 2001 2001
MEDIUM: print
CONFERENCE/MEETING: 43rd Annual Meeting of the American Society of Hematology, Part 1 Orlando, Florida, USA December 07-11, 2001; 20011207
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The in vivo administration of anti-CD40L mAb has been shown to promote alloengraftment under minimal or no pre-transplant conditioning in mice. In these studies, cyclosporine A (CsA) and rapamycin (rapa) were examined as single agents for their effects on alloengraftment and in combination with anti-**CD40L** mAb for their effects on anti-***CD40L*** -facilitated engraftment. CsA is a calcineurin inhibitor that blocks **T cell receptor** (**TCR**) signaling and prevents T cell activation and proliferation. Rapa inhibits IL-2 responsiveness, T cell proliferation and clonal expansion but does not interfere with T cell activation. We hypothesized that CsA, but not rapa, might interfere with anti-**CD40L**-facilitated alloengraftment since tolerance **induced** by costimulatory blockade requires activation of the ***TCR*** . Furthermore, since ***CD40L*** is upregulated only on activated CD4+ cells, but not CD8+ cells, rapa might be expected to increase engraftment in anti-CD40L mAb-treated mice by preventing the clonal expansion of potentially alloreactive CD8+ cells. C57BL/6 (H2b) mice received a minimally immune suppressive dose of 100 cGy TBI by x-ray on day -1 followed by a single infusion of a modest dose of 40X106 BALB/c (H2d) bone marrow cells on day 0. Irrelevant or anti-CD40L mAb was administered at 0.2 mg ip day -1 to 5, then twice weekly to day 14, +-CsA at 80 mg/kg day -1 to 13 or rapa at 1.5 mg/kg day -1 to 13. None of 30 mice receiving irrelevant mAb nor 20 mice receiving rapa as a single agent had evidence of donor chimerism by PBL typing at 6 weeks post transplant. CsA as a single agent resulted in only 3%+-7% average donor chimerism with 4/19 (21%) mice having detectable donor chimerism (gtoreq3% donor). In contrast, anti-CD40L mAb-treated mice had an average of 14%+-14% donor chimerism with 17/29 (59%) mice having detectable donor chimerism ($p<0.001$ vs irrelevant controls). Mice receiving anti-CD40L mAb+rapa had a dramatic increase in average donor chimerism to 40%+-5% donor with 20/20 (100%) mice having detectable donor chimerism ($p<0.001$ vs anti-CD40L as single agent). Surprisingly, CsA also facilitated engraftment in anti-CD40L mAb-treated mice. Mice receiving CsA+anti-CD40L mAb had an average of 35%+-6% donor chimerism with 20/20 (100%) mice

having detectable donor chimerism ($p<0.001$ vs anti-CD40L as single agent). Interestingly, although either CsA or rapa facilitated engraftment in anti-CD40L-treated mice, depletion of CD8 cells did not. Moreover, although CD8+ cells participate in the rejection process, CD8 depletion did not increase engraftment when combined with anti-CD40L as compared to anti-CD40L alone (13% vs 14%, $p=0.870$) suggesting that the increase in donor chimerism in anti-CD40L-treated mice by rapa or CsA was not due solely to their effects on CD8+ cells. Additional experiments revealed that rapa or CsA promoted equivalent high level of donor chimerism when combined with depleting anti-CD4 mAb rather than with anti-CD40L mAb even though depleting anti-CD4 mAb did not promote engraftment as a single agent. These data have important clinical ramifications and indicate that contrary to expectations, CsA and rapa may synergize with anti-CD40L mAb for the promotion of alloengraftment under nonmyeloablative conditions.

7/7/10 (Item 10 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013333273 BIOSIS NO.: 200100505112
N-substituted benzamides inhibit nuclear factor-kappaB and nuclear factor of activated T cells activity while inducing activator protein 1 activity in T lymphocytes
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JOURNAL: Molecular Immunology 38 (4): p267-277 August, 2001 2001
MEDIUM: print
ISSN: 0161-5890
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: N-substituted benzamides are compounds that have recently been reported to inhibit nuclear factor-kappaB (NF-kappaB) activity and induce apoptosis in a pre-B cell line. In this study, we focused on the effects of N-substituted benzamides on transcriptional regulation in Jurkat T cells. We used a model system where the cells can be stimulated either through TCR/CD28 or by treatment of the cells with PMA and ionomycin to induce transcription factors typical for T lymphocyte activation. Treatment of the Jurkat cells with procainamide did not influence the transcription factor profile of stimulated cells, while treatment with a derivative having an acetyl group in position 4 of the aromatic ring inhibited NF-kappaB and nuclear factor of activated T cells (NFAT) activity. Declopramide, which contains a chloride in position 3 of the aromatic ring, was inactive in this system, whereas also the acetylated derivative of this compound inhibited NF-kappaB and NFAT activity. In contrast, the transcriptional activity and nuclear expression of activator protein 1 **induced** by **TCR**/CD28 stimulation or PMA and ionomycin treatment was enhanced by the acetylated variants of the N-substituted benzamides. Finally, we investigated the effect of N-substituted benzamides on intact promoters for two genes central in immune regulation; the CD40 ligand (CD40L) and IL-2 promoters. The transcriptional activity of the **CD40L** promoter as well as surface expression of the **CD40L induced** by signaling through **TCR**/CD28 was inhibited by addition of acetylated N-substituted benzamides, while the transcriptional activity of the IL-2 promoter was enhanced. Taken together, these data indicate that derivatives of N-substituted benzamides are potential drug candidates for quantitative as well as qualitative modulation of immune functions.

7/7/11 (Item 11 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013106714 BIOSIS NO.: 200100278553
Role of CD40 ligand signaling in defective type-1 cytokine response in HIV infection
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JOURNAL: FASEB Journal 15 (4): pA308 March 7, 2001 2001
MEDIUM: print
CONFERENCE/MEETING: Annual Meeting of the Federation of American Societies for Experimental Biology on Experimental Biology 2001 Orlando, Florida, USA March 31-April 04, 2001; 20010331
ISSN: 0892-6638
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The pathogenesis of defective in vitro production of IL-12 and IFN-gamma (type-1 cytokine response) observed in HIV-infected patients, even those with high CD4 counts, remains to be fully elucidated. We investigated if perturbations in CD40L signaling are involved in the development of this defect. CD40 ligand trimer (CD40LT) stimulated PBMC production of IL-12 in response to Toxoplasma gondii and CMV. Regardless of CD4 count, CD40LT restored IL-12 secretion in response to T. gondii in HIV-infected patients. In the presence of CD40LT, PBMC from both HIV-infected patients and controls produced high levels of IL-12 in response to CMV. CD40LT restored T. gondii and CMV-triggered IFN-gamma secretion by T cells and PBMC from HIV-infected patients with CD4 counts > 200/mm3. The effect of CD40LT on IFN-gamma production was mediated through increased IL-12 secretion. Since CD40L regulates IL-12 production in response to T. gondii in humans, we examined if CD40L induction is impaired in HIV-infected patients. CD4+ T cells from these patients, even those with CD4 counts > 500/mm3, had defective **CD40L induction** after antigen presenting cell-mediated **TCR**/CD3 stimulation. Taken together, these results implicate impaired ***CD40L*** **induction** in the pathogenesis of defective IL-12/IFN-gamma production in HIV infection.

7/7/12 (Item 12 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0012029852 BIOSIS NO.: 199900289512
Functional and phenotypic analysis of thymic B cells: Role in the induction of T cell negative selection
AUTHOR: Ferrero Isabel; Anjuere Fabienne; Martin Pilar; del Hoyo Gloria Martinez; Lopez Fraga Marta; Wright Natalia; Varona Rosa; Marquez Gabriel ; Ardavin Carlos (Reprint)
AUTHOR ADDRESS: Department of Cell Biology, Faculty of Biology, Complutense University, E-28040, Madrid, Spain**Spain
JOURNAL: European Journal of Immunology 29 (5): p1598-1609 May, 1999 1999
MEDIUM: print
ISSN: 0014-2980
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The phenotype of mouse thymic B cells and their capacity to

induce T cell negative selection in vitro were analyzed. Thymic B cells expressed B cell markers such as IgM, Fcgamma receptor, CD44, heat-stable antigen, LFA-1 and CD40. In addition, they were positive for the activation molecule CD69 and displayed high levels of B7-2. Although thymic B cells expressed CD5 on their surface, no CD5-specific mRNA was detected. Moreover, thymic B cells ***induced*** a stronger deletion of **TCR**-transgenic (TG) thymocytes than splenic B cells, which had low CD69 and B7-2 levels. Interestingly, ***CD40*** -activated splenic B cells up-regulated CD69 and B7-2 and acquired a capacity to induce T cell deletion comparable to that of thymic B cells. Moreover, thymic B cells from CD40-deficient mice displayed lower CD69 and B7-2 levels than control thymic B cells, and lower capacity to **induce** the deletion of ***TCR*** TG thymocytes. These results support the hypothesis that **CD40**-mediated activation of thymic B cells determinesa high efficiency of antigen presentation, suggesting that within the thymus B cells may play an important role in the elimination of autoreactive thymocytes.

7/7/13 (Item 13 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0011960204 BIOSIS NO.: 199900219864
Regulation of CD40L expression by cyclic AMP: Contrasting proinflammatory and inhibitory actions
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JOURNAL: Cellular Immunology 192 (2): p203-212 March 15, 1999 1999
MEDIUM: print
ISSN: 0008-8749
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: CD40L expression is well recognized to be of critical importance in initiation of the immune response. Because cAMP mediates actions of bronchodilators commonly used in asthma, the effects of cAMP in regulating the immune response are of major importance. Cyclic AMP was found to either inhibit or markedly increase **CD40L** expression dependent upon the mechanisms of T cell activation. Cyclic AMP inhibited ***CD40L*** expression ***induced*** by ***TCR*** activation. In contrast, cAMP enhanced **CD40L induced** by CD2-mediated T cell activation or by calcium-dependent mechanisms. While neither CD28 costimulation nor exogenous IL-2 or IL-4 prevented cAMP inhibition in **TCR** activated cells, addition of calcium ionophore to **TCR** activation prevented any inhibitory effects and caused cAMP to increase ***CD40L*** expression. Actions of cAMP to increase ***CD40L*** expression appeared independent of PKC and were not a reflection of generalized cellular activation since neither CD25 nor CD69 expression was affected. The markedly contrasting actions of cAMP to decrease or increase CD40L expression, an important control point in the immune response, could be relevant to actions of commonly used medications including bronchodilators.

7/7/14 (Item 14 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0011553658 BIOSIS NO.: 199800347905
Th1 cells induce and Th2 inhibit antigen-dependent IL-12 secretion by dendritic cells

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JOURNAL: European Journal of Immunology 28 (6): p2003-2016 June, 1998 1998
MEDIUM: print
ISSN: 0014-2980
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Dendritic cells are the most relevant antigen-presenting cells (APC) for presentation of antigens administered in adjuvant to CD4+ T cells. Upon interaction with antigen-specific T cells, dendritic cells (DC) expressing appropriate peptide-MHC class II complexes secrete IL-12, a cytokine that drives Th1 cell development. To analyze the T cell-mediated regulation of IL-12 secretion by DC, we have examined their capacity to secrete IL-12 in response to stimulation by antigen-specific Th1 and Th2 D011.10 ***TCR*** -transgenic cells. These cells do not differ either in **TCR** clonotype or **CD40 ligand (CD40L)** expression. Interaction with antigen specific Th1, but not Th2 cells, induces IL-12 p40 and p75 secretion by DC. The induction of IL-12 production by Th1 cells does not depend on their IFN-gamma secretion, but requires direct cell-cell contact mediated by peptide/MHC class II-***TCR*** and ***CD40*** - ***CD40L*** interactions. Th2 cells not only fail to **induce** IL-12 secretion, but they inhibit its induction by Th1 cells. Unlike stimulation by Th1, inhibition of IL-12 production by Th2 cells is mediated by soluble molecules, as demonstrated by transwell cultures. Among Th2-derived cytokines, IL-10, but not IL-4 inhibit Th1-driven IL-12 secretion. IL-10 produced by Th2 cells appears to be solely responsible for the inhibition of Th1-induced IL-12 secretion, but it does not account for the failure of Th2 cells to induce IL-12 production by DC. Collectively, these results demonstrate that Th1 cells up-regulate IL-12 production by DC via IFN-gamma-independent cognate interaction, whereas this is inhibited by Th2-derived IL-10. The inhibition of Th1-induced IL-12 production by Th2 cells with the same antigen specificity represents a novel mechanism driving the polarization of CD4+ T cell responses.

7/7/15 (Item 15 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0011525330 BIOSIS NO.: 199800319577
A humanised therapeutic CD4 mAb inhibits **TCR-induced** IL-2, IL-4, and IL-10 secretion and expression of CD25, **CD40L**, and CD69
AUTHOR: Woods Margaret (Reprint); Guy Robert (Reprint); Waldmann Herman; Glennie Martin; Alexander Denis R (Reprint)
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JOURNAL: Cellular Immunology 185 (2): p101-113 May 1, 1998 1998
MEDIUM: print
ISSN: 0008-8749
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The actions of a humanised therapeutic CD4 mAb YHB.46 on T cell activation were investigated in vitro. Soluble YHB.46 IgG or YHB.46-derived F(ab')2 fragments caused inhibitions of up to 100% of the proliferation of purified CD4+ T cells activated with immobilised CD3 mAb. The inhibitory effects of the CD4 mAb were equally potent in both CD45RA+ and CD45RO+ T cell subset proliferation assays. Inhibitory effects on DNA synthesis were not explicable by increased T cell

apoptosis. YHB.46 was inhibitory even when added 70 h after exposure of cells to immobilised CD3 mAb, but it had little effect on IL-2 receptor-driven proliferation signals. The CD4 mAb inhibited the CD3-induced expression of the CD25 and CD69 activation markers on the T cell surface and suppressed CD40 ligand expression, but not that of CD25 and CD69, when their expression was induced by phorbol ester plus ionomycin. YHB.46 also exerted a profound inhibitory effect on the production of IL-2, IL-4, and IL-10, irrespective of whether T cells were activated with CD3 mAb or with phorbol ester plus ionomycin. The inhibitory effects of YHB.46 on CD4+ T cell proliferation were partially prevented by the addition of exogenous IL-2 or autologous monocytes and were completely prevented by activating T cells with a novel CD3-CD28 bivalent F(ab')2 reagent. However, the inhibitory effects of YHB.46 on T cell proliferation were equipotent in the presence or the absence of CTLA-4Ig, showing that the CD4 mAb was not acting on CD28-induced activation signals per se. Our results show that the inhibitory effects of YHB.46 on T cell activation do not involve CD28 or IL-2 receptor signalling, but are directed at the TCR-mediated G0-G1 transition. These findings in vitro predict that YHB.46 may act as a potent immunosuppressant in the clinical context.

7/7/16 (Item 16 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0010464429 BIOSIS NO.: 199699098489
Functional **CD40 ligand** expression on T lymphocytes in the absence of **T cell receptor** engagement: Involvement in interleukin-2-**induced** interleukin-12 and interferon-gamma production
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JOURNAL: European Journal of Immunology 26 (7): p1430-1434 1996 1996
ISSN: 0014-2980
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Despite the fact that the great majority of T cells at the site of an inflammatory response are not antigen specific, the mechanisms leading to activation and recruitment of these bystander T cells are poorly understood. We previously reported that soluble (s)CD23 potentiated the interleukin (IL)-2-induced interferon (IFN)-gamma production by T cells co-cultured with autologous monocytes in the absence of ***T*** ***cell*** ***receptor*** (***TCR***) engagement. Our present data demonstrate that the IL-2-**induced** IFN-gamma secretion, in the presence but also in the absence of sCD23, is strictly IL-12 dependent, inasmuch as anti-IL-12 antibody abrogated both responses. Most interestingly, anti-CD40 ligand (CD40L) monoclonal antibody significantly inhibited IL-2-induced IL-12 as well as IFN-gamma production. These results suggest that **CD40L** was expressed on T cells in the absence of ***TCR*** engagement. Indeed, purified unstimulated T cells readily expressed ***CD40L*** . IL-2 and monocytes did not up-regulate ***CD40L*** on resting T cells. It is proposed that low levels of CD40L expression on non-antigen stimulated T cells are sufficient to signal through CD40 molecules on accessory cells and to induce IL-12 secretion, which in turn can synergize with IL-2 for the induction of IFN-gamma production, thus contributing to the inflammatory process.

7/7/17 (Item 17 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0010276433 BIOSIS NO.: 199698744266
Regulation of **CD40 ligand** expression on naive CD4 T cells: A role for **TCR** but not co-stimulatory signals
AUTHOR: Jaiswal Archana I; Dubey Caroline; Swain Susan L; Croft Michael (Reprint)
AUTHOR ADDRESS: Cancer Center, Univ. California San Diego, 9500 Gilman Dr., La Jolla, CA 92093-0063, USA**USA
JOURNAL: International Immunology 8 (2): p275-285 1996 1996
ISSN: 0953-8178
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: We have investigated the roles of **TCR** and accessory co-stimulatory signals in the **induction** of **CD40 ligand** (***CD40L***) on CD4 cells. Using naive T cells from ***TCR*** transgenic mice, specific for a peptide of pigeon cytochrome c, we show that in contrast to IL-2 secretion, **CD40L** expression is regulated primarily by signaling through the **TCR**, is enhanced by accessory molecule interactions, but co-stimulatory signals play little if any role. **CD40L** was induced at high levels on naive T cells, peaking at 5 h, by class II MHC+ fibroblast antigen-presenting cells (APC) which expressed either ICAM-1, B7-1 or both molecules, whereas only low levels were induced by fibroblasts which did not express any accessory molecules. Differences in intensity and duration of expression were seen following stimulation with ICAM- and B7-expressing APC, with the presence of ICAM resulting in greater and longer expression, although both molecules together were most efficient. The involvement of co-stimulatory signals delivered from accessory molecules was investigated in systems where there was no effect on TCR signaling from adhesive interactions. Anti-CD3, or antigen-pulsed APC lacking accessory molecules, were used to provide the TCR signal, with costimulus from either anti-CD28 or accessory molecule-expressing fibroblasts not presenting antigen. Anti-CD3 in the absence of co-stimuli induced high CD40L expression but no IL-2 production and provision of co-stimulatory signals, although inducing large quantities of IL-2, did not increase CD40L expression. In addition, low CD40L expression induced by antigen presented in the absence of accessory molecules was not enhanced by co-stimulation, although IL-2 was strongly up-regulated. These studies suggest that efficient expression of CD40L on naive CD4 cells does require accessory molecules on APC. However, the role of these molecules for CD40L induction, as opposed to IL-2 secretion, is not one of co-stimulation but one of adhesion, presumably allowing stronger or more prolonged signals to be generated through the TCR. The synergistic role of ICAM and B7 during naive CD4 activation was confirmed using dendritic cells as APC, with nearly complete inhibition of CD40L expression as well as IL-2 secretion being seen when both CTLA-4-Ig and anti-LFA-1 were used to block these molecules.

7/7/18 (Item 18 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0010260383 BIOSIS NO.: 199698728216
Parietaria judaica-specific T-cell clones from atopic patients: Heterogeneity in restriction, V-beta usage, and cytokine profile
AUTHOR: Sallusto Federica; Corinti Silvia; Pini Carlo; Biocca Marina M; Bruno Guglielmo; Felice Gabriella Di (Reprint)

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JOURNAL: Journal of Allergy and Clinical Immunology 97 (2): p627-637 1996 1996
ISSN: 0091-6749
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The pollen of Parietaria spp. is one of the most clinically relevant sources of allergens in the Mediterranean area. CD4+ T-lymphocyte clones specific for Parietaria allergens were isolated from peripheral blood of atopic donors, and their phenotype, HLA restriction, V-beta usage, and cytokine profile were determined. All the T-cell clones expressed the alpha/beta **T-cell receptor** and were **induced** to express **CD40 ligand** after activation with phorbol-myristate-acetate plus ionomycin. When the proliferative response to three chromatographic fractions of the extract was analyzed, distinct reactivity patterns were found. Interestingly, most of the clones responded to the fraction that was the most enriched for the major allergen Par j 1. The clones were either HLA-DR- or HLA-DQ-restricted and did not show any preferential usage of T-cell receptor V-beta segments. Five of the 17 clones tested produced only IL-4 and no interferon-gamma, thus displaying a T-H2 phenotype. The other clones displayed a T-H0 phenotype in that they produced both IL-4 and interferon-gamma. These results show that in atopic patients T-cell response against Parietaria judaica allergen involves different T-cell subsets in terms of restriction, V-beta usage, and cytokine profile.

7/7/19 (Item 19 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0010095661 BIOSIS NO.: 199698563494
Altered CD40 ligand induction in tolerant T lymphocytes
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JOURNAL: European Journal of Immunology 25 (10): p2830-2834 1995 1995
ISSN: 0014-2980
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: CD40 ligand (CD40L) is a member of the tumor necrosis factor superfamily and is expressed on the surface of activated T lymphocytes. The interaction of CD40L with CD40 on B cells results in B cell activation, immunoglobulin (IG) secretion and Ig class switching. To study anergy as a mechanism of murine CD4 T-cell tolerance, we determined both in vivo and in vitro that CD3-activated anergic cells are deficient in the ability to stimulate B cell proliferation, and that anergic cells are defective for the **T cell receptor**/CD3-mediated ***induction*** of ***CD40L*** expression. These results have implications for the recruitment of B cell responses by anergic T cells in vivo.

7/7/20 (Item 20 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0009203005 BIOSIS NO.: 199497224290
CD40 ligand acts as a costimulatory signal for neonatal thymic gamma-delta

T cells
AUTHOR: Ramsdell Fred (Reprint); Seaman Michael S; Clifford K N; Fanslow William C
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JOURNAL: Journal of Immunology 152 (5): p2190-2197 1994 1994
ISSN: 0022-1767
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The stimulatory requirements for T cells bearing gamma-delta **T cell receptors** are distinct from those of alpha-beta T cells. We have analyzed the ability of the ***CD40*** ***ligand*** (***CD40L***) to activate neonatal thymic gamma-delta T cells. CD40L is expressed on activated T cells and has been shown to induce B cell proliferation and Ig secretion as well as monocyte activation. We now demonstrate that, in the presence of an anti-**TCR**-gamma-delta Ab, **CD40L** is able to **induce** the proliferation of neonatal thymic gamma-delta cells. The presence of ***CD40L*** also leads to enhanced expression of a variety of activation-associated Ag including CD25, CD69, CD44, and Ly6C. In addition to proliferation, CD40L induces lectin-mediated cytolytic activity in thymic gamma-delta T cells as well as the production of IFN-gamma and TNF-alpha. We were unable to detect IL-2 or IL-4 production in response to CD40L, and Ab-blocking studies indicate that the mechanism of activation appears to involve IL-1 but is independent of IL-2, IL-4, and IL-7. These results suggest that, in addition to its effects on B cells and monocytes, CD40L can costimulate the activation of thymic gamma-delta T cells.

7/7/21 (Item 21 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0009053879 BIOSIS NO.: 199497075164
Ligation of B7 with CD28/CTLA-4 on T cells results in CD40 ligand expression, interleukin-4 secretion and efficient help for antibody production by B cells
AUTHOR: De Boer Mark (Reprint); Kasran Ahmad; Kwekkeboom Jaap; Walter Hugo; Vandenberghe Peter; Ceuppens Jan L
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JOURNAL: European Journal of Immunology 23 (12): p3120-3125 1993 1993
ISSN: 0014-2980
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: It has been extensively shown that when T cells are co-stimulated with B7-CD28 interaction, a strong proliferative as well as cytolytic T cell response can be induced. In contrast, there exists only indirect evidence that the B7-CD28 interaction is of importance for the induction of T cell helper functions in B cell responses. Here we have used mouse fibroblasts transfected with the human Fc-gamma receptor type II and human B7 to address this issue. We found that T cells, when activated through the T cell receptor (TcR)/CD3 complex with monoclonal antibodies and co-stimulated by B7-CD28 interaction, can provide efficient help for the induction of both IgM and IgG production by resting B cells. This helper activity is, at least in part, mediated by the interaction between the CD40 ligand on the T cells and CD40 on the B cells. We also demonstrate that more than one signal to the T cell is required for the **induction** of the **CD40 ligand**, one being delivered through the **TcR**/CD3 complex and the second by ligation of CD28 with

the B7 molecule. In addition to the induction of cognate T helper function, we provide evidence that co-stimulation of T cells with B7-CD28 interaction can result in the secretion of both Th1- and Th2-type lymphokines.

7/7/22 (Item 22 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0008210564 BIOSIS NO.: 199293053455
TUMOR NECROSIS FACTOR TNF RECEPTOR EXPRESSION IN T LYMPHOCYTES DIFFERENTIAL REGULATION OF THE TYPE I TNF RECEPTOR DURING ACTIVATION OF RESTING AND EFFECTOR T CELLS
AUTHOR: WARE C F (Reprint); CROWE P D; VANARSDALE T L; ANDREWS J L; GRAYSON M H; JERZY R; SMITH C A; GOODWIN R G
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JOURNAL: Journal of Immunology 147 (12): p4229-4238 1991
ISSN: 0022-1767
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: ENGLISH

ABSTRACT: The expression of TNF-α receptors (TNFR) was examined on a CD4+ T cell hybridoma, transformed T cell lines, CTL clones, and activated T cells from peripheral blood to determine the basis of the immunomodulatory activity of TNF on T cell function. Analyses by ligand cross-linking and competitive binding assays with mAb to the 80-kDa receptor (TNFR-I), demonstrated that the TNFR-I was the predominant receptor expressed on activated CD4+ and CD8+ T cell subsets. However, on T cell leukemic lines, a second, non-TNFR-I binding site was identified, most likely the 55-kDa form (TNFR-II). Additional subsets of T cells were readily distinguished by their expression of TNFR-I and related members of the TNFR gene family (***CD40*** and CD27). Expression of the TNFR-I was dependent upon the state of T cell activation. Signaling through the **TCR** for Ag or IL-2R was sufficient to **induce** TNFR mRNA and protein expression in resting T cells. Multiple sizes of TNFR-I transcripts were detected during T cell activation; however, biosynthetic studies showed these multiple species encode a single protein of 80 kDa. These results, combined with the known ability of TNF to induce IL-2R expression, indicate that TNF and IL-2 form a reciprocating receptor amplification circuit. In contrast, differentiated effector T cells triggered through the TCR or protein kinase C initiated a rapid down-regulation (transmodulation) of the TNFR-I that preceded TNF or lymphotoxin secretion. The mechanism of transmodulation involved proteolytic processing of the mature 80-kDa receptor releasing a soluble 40-kDa fragment. This indicates that a TNF autocrine loop is not likely to form during the response of an effector T cell. Collectively, these results suggest that transcriptional and post-translational modification of the TNFR-I are important control points regulating the expression of this receptor during T cell activation.

7/7/23 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

12019226 EMBASE No: 2003129970
Cutting edge: CD40-induced expression of recombination activating gene (RAG) 1 and RAG2: A mechanism for the generation of autoaggressive T cells in the periphery
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Journal of Immunology (J. IMMUNOL.) (United States) 01 APR 2003, 170/7 (3455-3459)
CODEN: JOIMA ISSN: 0022-1767
DOCUMENT TYPE: Journal ; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 24

It has been speculated that autoimmune diseases are caused by failure of central tolerance. However, this remains controversial. We have suggested that CD40 expression identifies autoaggressive T cells in the periphery of autoimmune prone mice. In this study, we report that CD40 was cloned from autoaggressive T cells and that engagement induces expression and nuclear translocation of the recombinases, recombination activating gene (RAG) 1 and RAG2 in the autoaggressive, but not in the nonauto-aggressive, peripheral T cell population. Furthermore, we demonstrate that ***CD40*** engagement **induces** altered **TCR** Valpha, but not Vbeta, expression in these cells. Therefore, ***CD40*** -regulated expression of RAG1 and RAG2 in peripheral T cells may constitute a novel pathway for the generation of autoaggressive T cells.

7/7/24 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE

11606223 EMBASE No: 2002170539
Sustained NFAT signaling promotes a Th1-like pattern of gene expression in primary murine CD4SUP+ T cells
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Journal of Immunology (J. IMMUNOL.) (United States) 15 MAY 2002, 168/10 (4936-4945)
CODEN: JOIMA ISSN: 0022-1767
DOCUMENT TYPE: Journal ; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 64

T cell activation is known to be critically regulated by the extent and duration of ***TCR*** - ***induced*** signaling pathways. The NFAT family of transcription factors is believed to play an important role in coupling these quantitative differences in **TCR-induced** signaling events into changes in gene expression. In this study we have specifically investigated the effects of sustained NFAT signaling on T cell activation by introducing a constitutively active mutant version of NFATc1 (caNFATc1) into primary murine CD4SUP+ T cells and examining its effects on gene expression. We now report that ectopic expression of caNFATc1 partially mimics **TCR** signaling, resulting in enhanced expression of CD25 and ***CD40*** ***ligand*** and down-regulation of CD62L. More importantly, we find that expression of caNFATc1 in T cells maintained under either nonpolarizing or Th1-skewing conditions leads to a marked selective increase in the number of cells expressing the prototypical Th1 cytokine, IFN-gamma. Furthermore, when expressed in Th2-skewed cells, caNFATc1 appears to attenuate Th2 differentiation by decreasing production of IL-4 and promoting the expression of IFN-gamma. Finally, we find that caNFATc1 enhances expression of functional P-selectin glycoprotein ligand-1, up-regulates Fas ligand expression, and increases susceptibility to activation-induced cell death, cellular traits that are preferentially associated with Th1 effector cells. Taken together, these results suggest

that sustained NFAT signaling, mediated by ectopic expression of caNFATc1, acts to promote a Th1-like pattern of gene expression and thereby serves to highlight the important relationship between the degree of NFAT signaling and the qualitative pattern of gene expression induced during T cell activation.

7/7/25 (Item 3 from file: 73)
DIALOG(R)File 73:EMBASE

11101671 EMBASE No: 2001112786
Regulation of transcriptional activity of the murine **CD40 ligand** promoter in response to signals through **TCR** and the costimulatory molecules CD28 and CD2
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Journal of Immunology (J. IMMUNOL.) (United States) 01 APR 2001, 166/7 (4578-4585)
CODEN: JOIMA ISSN: 0022-1767
DOCUMENT TYPE: Journal ; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 56

We have analyzed the murine CD40 ligand promoter with regard to stimulation of transcriptional activity in Jurkat T cells after signaling via the ***TCR*** and the costimulatory molecules CD28 and CD2. ***TCR*** engagement was necessary for the **induction** of transcriptional activity from the **CD40 ligand** promoter, and costimulation through either CD28 or CD2 further increased the activity. Analysis of promoter deletants showed that the DNA elements needed for transcriptional activity induced by costimulatory molecules were located within two regions containing previously identified transcription factor NFAT sites. Further studies of the proximal NFAT site showed that it was not dependent on AP-1 binding for transcriptional activity induced by costimulation through CD28. Instead, a region between the TATA box and the proximal NFAT site was shown to bind proteins of the early growth response family and to contribute to NFAT-mediated transcriptional activation.

7/7/26 (Item 4 from file: 73)
DIALOG(R)File 73:EMBASE

11062183 EMBASE No: 2001078874
Identification of a CD28 response element in the CD40 ligand promoter
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Dr. E. Parra, Sidney Kimmel Cancer Center, 10835 Altman Row, San Diego, CA 92121 United States
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Journal of Immunology (J. IMMUNOL.) (United States) 15 FEB 2001, 166/4 (2437-2443)
CODEN: JOIMA ISSN: 0022-1767
DOCUMENT TYPE: Journal ; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 49

Ligation of the T cell coreceptor CD28 or CD2 by its cognate ligands B7-1 or LFA-3, respectively, greatly aids the Ag-induced up-regulation of several genes, including IL-2 and CD40 ligand (CD40L). Using luciferase reporter constructs under the control of the 1.2 kb of 5 noncoding region of the human CD40L gene, we have found that stimulation through CD28 was

required for a strong transcriptional activity of the **CD40L** promoter in response to **TCR** ligation, while the activity **induced** by CD2 was slightly lower than CD28. Deletion analysis demonstrated that the transcriptional elements mediating this effect were located within a 300-bp region upstream of the start site. Further dissection of this region and gel shift analyses demonstrated the presence of a CD28 response element in a region located between nucleotides -170 to -164 relative to the start site. Transcriptional studies with a CD40L enhancer-promoter carrying a mutation in this putative CD28 response element revealed that the activity was reduced by 80 and 70% after B7-1 and LFA-3 costimulation, respectively. The transcription factor complex bound to this site contained at least JunD, c-Fos, p50, p65, and c-Rel, but not c-Jun. Mutations introduced into the CD28RE also blocked the binding of this complex. These observations identify an important role for the CD28 signaling pathway in the regulation of CD40L promoter transcriptional activity.

7/7/27 (Item 5 from file: 73)
DIALOG(R)File 73:EMBASE

11035624 EMBASE No: 2001048816
Broad programming by IL-2 receptor signaling for extended growth to multiple cytokines and functional maturation of antigen-activated T cells
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Journal of Immunology (J. IMMUNOL.) (United States) 01 FEB 2001, 166/3 (1675-1683)
CODEN: JOIMA ISSN: 0022-1767
DOCUMENT TYPE: Journal ; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 46

Coincident production of IL-2 and **induction** of high-affinity IL-2R upon **TCR** engagement has precluded a clear distinction for the biological outcome of signaling through TCR/costimulatory molecules vs the IL-2R. Using a novel transgenic mouse on the IL-2RbetaSUP-/- genetic background, this study has separated the relative outcome of signaling through the ***TCR*** and IL-2R. We show that stimulation through the **TCR** and CD28 or **CD40 ligand** directly leads to T cell activation and several rounds of proliferation in an IL-2-independent fashion. However, this stimulation is insufficient for extended T cell growth to multiple cytokines or differentiation into CTL or IFN-gamma-secreting effector T cells. IL-2 is required for these functions in part by regulation of cyclin D3 and granzyme B. Somewhat less efficiently, IL-4 stimulation of these transgenic T cells redundantly rescued many of these activities. These data demonstrate a fundamental requirement for IL-2 and perhaps other common gamma-chain-dependent cytokines to promote selective gene expression by Ag-activated T cells for their subsequent growth and differentiation into effector T lymphocytes.

7/7/28 (Item 6 from file: 73)
DIALOG(R)File 73:EMBASE

07749049 EMBASE No: 1999231072
CD40-CD154 interaction and IFN-gamma are required for IL-12 but not prostaglandin Einf 2 secretion by microglia during antigen presentation to Th1 cells
Aloisi F.; Penna G.; Polazzi E.; Minghetti L.; Adorini L.

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Journal of Immunology (J. IMMUNOL.) (United States) 01 FEB 1999, 162/3 (1384-1391)
CODEN: JOIMA ISSN: 0022-1767
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 58

IL-12 and PGEinf 2 promote and inhibit, respectively, the development of Th1 responses. Production of these mediators by APC residing in the central nervous system (CNS) may be involved in the local regulation of the T cell phenotype during infectious and autoimmune CNS diseases. In the present study we have examined IL-12 and PGEinf 2 secretion by cultured microglia and astrocytes from the mouse brain upon Ag-dependent interaction with I-A(d)- restricted, OVAsup 3sup 2sup 3sup -sup 3sup 3sup 9 specific TCR transgenic Th1 and Th2 cell lines. We show that microglia, which restimulate efficiently both Th1 and Th2 cells, secrete IL-12 upon Ag-dependent interaction with Th1, but not with Th2 cells. Th1-driven IL-12 production depends on **TCR** ligation by MHC class II/peptide complexes, **CD40** engagement on microglia, and IFN-gamma, secretion by activated Th1 cells. Th1 and, to a lesser extent, Th2 cells also stimulate the production of PGEinf 2 by microglia. T cell-mediated ***induction*** of PGEinf 2 requires MHC class II/peptide/**TCR** interactions but does not depend on ***CD40*** engagement or on the presence of IFN-gamma. Astrocytes, which preferentially activate Th2 cells, fail to produce IL-12 and secrete negligible amounts of PGEinf 2 upon interaction with either Th1 or Th2 cells. These results suggest that during CNS infection or immunopathology, IL-12 produced by microglia upon Ag- specific interaction with Th1 cells may further skew the immune response to Th1, whereas the T cell-dependent production of PGEinf 2 by microglia may represent a negative feedback mechanism, limiting the propagation of Th1 responses.

7/7/29 (Item 7 from file: 73)
DIALOG(R)File 73:EMBASE

07740269 EMBASE No: 1999217790
TCR vaccines against T cell lymphoma: QS-21 and IL-12 adjuvants induce a protective CD8sup + T cell response
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Journal of Immunology (J. IMMUNOL.) (United States) 15 FEB 1999, 162/4 (2251-2258)
CODEN: JOIMA ISSN: 0022-1767
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 50

Tumor-specific TCR can serve as an effective target for active immunotherapy of T cell malignancies. Using the murine T cell tumor model C6VL, vaccination with C6VL TCR protected mice from a subsequent lethal dose of tumor cells. This study characterizes the immune mechanisms involved in the tumor protection, and the influence of immunologic adjuvants in ***inducing*** a protective immune response. Immune responses **induced** by **TCR** vaccines formulated with various adjuvants: QS-21, IL-12, SAF-1, ***CD40L*** , and GM-CSF were compared. QS-21, IL-12, and SAF-1 biased the humoral immune response toward Th1-type, reflected by the ***induction*** of IgG2a and IgG2b anti-C6VL ***TCR*** Absolute ***CD40L***

and GM-CSF exclusively produced IgG1 Abs, reflecting a Th2-type immune response. In our tumor model system, only vaccines containing adjuvants that induced a Th1-type immune response favored tumor protection. Furthermore, we demonstrated that CD8sup + T cells were necessary and sufficient for tumor protection using anti-CD8 mAb depletion and adoptive cell transfer experiments. Transfer of hyperimmune serum containing anti-C6VL TCR Abs into naive mice had modest anti-tumor effects and was not sufficient to prevent tumor growth. TCR-vaccinated B cell-deficient mice were not protected against C6VL tumor, and tumor protection was not completely restored after hyperimmune serum transfer. Thus, B cells may serve as important APCs in ***inducing*** a protective immune response. Based on these results future **TCR** vaccines should be designed to maintain native **TCR** conformation, as well as **induce** a strong Th1-type immune response.

7/7/30 (Item 8 from file: 73)
DIALOG(R)File 73:EMBASE

07411640 EMBASE No: 1998313042
Signaling through a CD3gamma-deficient TCR/CD3 complex in immortalized mature CD4sup + and CD8sup + T lymphocytes
Pacheco-Castro A.; Alvarez-Zapata D.; Serrano-Torres P.; Regueiro J.R.
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Journal of Immunology (J. IMMUNOL.) (United States) 15 SEP 1998, 161/6 (3152-3160)
CODEN: JOIMA ISSN: 0022-1767
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 48

The biologic role of each CD3 chain and their relative contribution to the signals transduced through the TCR/CD3 complex and to downstream activation events are still controversial: they may be specialized or redundant. We have immortalized peripheral blood CD4sup + and CD8sup + T lymphocytes from a human selective CD3gamma deficiency using Herpesvirus saimiri. The accessibility of the mutant TCR/CD3 complex to different Abs was consistently lower in immortalized CD8sup + cells when compared with CD4sup + cells, relative to their corresponding CD3gamma-sufficient controls. Several ***TCR*** /CD3- ***induced*** downstream activation events, immediate (calcium flux), early (cytotoxicity and induction of surface CD69 or **CD40L** activation markers or intracellular TNF-alpha) and late (proliferation and secretion of TNF-alpha), were normal in gamma-deficient cells, despite the fact that their TCR/CD3 complexes were significantly less accessible than those of controls. In contrast, the accumulation of intracellular IL-2 or its secretion after CD3 triggering was severely impaired in gamma-deficient cells. The defect was upstream of protein kinase C activation because addition of transmembrane stimuli (PMA plus calcium ionophore) completely restored IL-2 secretion in gamma-deficient cells. These results suggest that the propagation of signals initiated at the TCR itself can result in a modified downstream signaling cascade with distinct functional consequences when gamma is absent. They also provide evidence for the specific participation of the CD3gamma chain in the induction of certain cytokine genes in both CD4sup + and CD8sup + human mature T cells. These immortalized mutant cells may prove to be useful in isolating cytosolic signaling pathways emanating from the TCR/CD3 complex.

7/7/31 (Item 9 from file: 73)
DIALOG(R)File 73:EMBASE

07339520 EMBASE No: 1998235639
Anti-CD40L accelerates renal disease and adenopathy in MRL-lpr mice in parallel with decreased thymocyte apoptosis
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Journal of Immunology (J. IMMUNOL.) (United States) 15 JUL 1998, 161/2 (729-739)
CODEN: JOIMA ISSN: 0022-1767
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 49

The CD40/CD40L (CD40 ligand) axis regulates several interactions between T cells and B cells. Blocking of CD40 engagement by CD40L inhibits Ig class switch by B cells as well as diminishes T cell response to an immunizing Ag. For these reasons, disruption of CD40/CD40L interactions by anti-CD40L administration or by genetic disruption of CD40L has ameliorated a variety of autoimmune conditions. More recent findings suggest that a direct signal can be transmitted to T cells via their expressed CD40L, which can costimulate proliferation with CD3 or promote germinal center formation. It is therefore possible that treatment with anti-CD40L Ab might produce a different outcome than observed in genetically CD40L-deficient mice. In this regard, we observe that in contrast to the genetic deletion of CD40L in MRL-lpr mice, which diminishes autoimmune disease but has little effect on adenopathy, administration of anti-CD40L to MRL-lpr mice accelerates both of these parameters. This difference appears to result from anti-CD40L actively delivering a signal that inhibits T cell apoptosis in lpr mice. This was confirmed by in vitro studies demonstrating that **CD40L** cross-linking on lpr thymocytes inhibited apoptosis and surface **TCR** down-modulation ***induced*** by CD3 ligation.

7/7/32 (Item 10 from file: 73)
DIALOG(R)File 73:EMBASE

06403974 EMBASE No: 1996067029
Parietaria judaica-specific T-cell clones from atopic patients: Heterogeneity in restriction, Vbeta usage, and cytokine profile
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Journal of Allergy and Clinical Immunology (J. ALLERGY CLIN. IMMUNOL.) (United States) 1996, 97/2 (627-637)
CODEN: JACIB ISSN: 0091-6749
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

The pollen of Parietaria spp. is one of the most clinically relevant sources of allergens in the Mediterranean area. CD4sup + T-lymphocyte clones specific for Parietaria allergens were isolated from peripheral blood of atopic donors, and their phenotype, HLA restriction, Vbeta usage, and cytokine profile were determined. All the T-cell clones expressed the alpha/beta **T-cell receptor** and were **induced** to express **CD40 ligand** after avtivation with phorbol-myristate-acetate plus ionomycin. When the proliferative response to three chromatographic fractions of the extract was analyzed, distinct reactivity patterns were found. Interestingly, most of the clones responded to the fraction that was the most enriched for the major allergen Par j 1. The clones were either HLA-DR- or HL4-DQ-restricted and did not show any

preferential usage of T-cell receptor Vbeta segments. Five of the 17 clones tested produced only IL-4 and no interferon-gamma, thus displaying a T(H2) phenotype. The other clones displayed a T(H0) phenotype in that they produced both IL-4 and interferon-gamma. These results show that in atopic patients T- cell response against Parietaria judaica allergen involves different T-cell subsets in terms of restriction, Vbeta usage, and cytokine profile.

7/7/33 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

14484096 PMID: 10481375
[Regulation of the contact sensitivity reaction by suppression of T gamma delta lymphocytes]
Regulacja reakcji nadwrazliwosci kontaktowej przez supresyjne limfocyty T gamma delta.
Szczepanik M
Katedra Immunologii Collegium Medicum UJ w Krakowie.
Folia medica Cracoviensia (POLAND) 1998, 39 (1-2) p5-33, ISSN 0015-5616 Journal Code: 0374617
Document type: Journal Article; Review; Review, Tutorial ; English Abstract
Languages: POLISH
Main Citation Owner: NLM
Record type: Completed
Contact sensitivity (CS) is a classical example of in vivo T cell mediated immune response that is under regulation. It is well known that in normal mice suppression of CS can be mediated by T alpha beta cells tolerized by prior exposure to high dose of antigen (Ag). In this paper it was shown that treatment of defective TCR alpha -/- or **TCR** beta -/- mice with high dose of Ag may result in **induction** of T gamma delta suppressor cells, that are able to inhibit both adoptive cell transfer of CS in vivo and IFN-gamma production in vitro. These suppressor cells are characterized as: **TCR** gamma delta+, CD3+, CD4-, CD8-, CD28+, ***CD40L*** +, CD95 (Fas)+, Fc gamma R+ and NK1.1-. Suppression mediated by T gamma delta cells showed antigen specificity, but was not restricted by the MHC. T gamma delta suppressor cells express very strong down-regulatory activity where even 2.5 x 10(3) T gamma delta cells could suppress 7 x 10(7) CS-effector cells. Presented data may suggest that IL-4 released by T gamma delta suppressor cells is involved in the mechanism of their down-regulatory function. (68 Refs.)
Record Date Created: 19990928
Record Date Completed: 19990928

7/7/34 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

14430614 PMID: 10425271
Increased expression of **CD40** on thymocytes and peripheral T cells in autoimmunity: a mechanism for acquiring changes in the peripheral ***T*** ***cell*** ***receptor*** repertoire.
Wagner D H; Newell E; Sanderson R J; Freed J H; Newell M K
Webb-Waring Institute for Cancer, Aging and Antioxidant Research, Denver, CO 80262, USA.
International journal of molecular medicine (GREECE) Sep 1999, 4 (3) p231-42, ISSN 1107-3756 Journal Code: 9810955
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

CD40, a cell surface molecule found on B lymphocytes and other antigen presenting cells, can, when engaged by **CD40 ligand** (**CD40L**), induce gene ***rearrangements*** and isotype switching. We report here that **CD40** is also expressed on thymocytes and on up to 50% of peripheral T cells from autoimmune prone strains of mice. In normal animals, CD40 is present on a small population of T cells and thymocytes. CD40 is expressed on most T cell hybridomas. We demonstrate that CD40 engagement on peripheral T cells, T cell hybridomas and thymocytes results in altered TCRValpha expression. That induced expression of different Valpha's results from the activity of the recombinase gene is implied by the observation that **CD40** does not **induce TCR** changes in RAG knock-out mice. Total cell numbers remained unchanged between anti-**CD40** treated and untreated populations of thymocytes or T cells indicating that treatment does not induce cell proliferation or cell death. The data presented here suggest a mechanism by which self reactive T cells accumulate peripherally and independently of selective processes of the thymus.

Record Date Created: 19990916
Record Date Completed: 19990916

7/7/35 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

14263946 PMID: 10083602

CD40 upregulation in **TCR** alpha/beta+ CD68+ cells and parenchymal **CD40L induction** and associated with NF-kappa B activation in chronic rejecting human renal allografts.

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Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Record Date Created: 19990415
Record Date Completed: 19990415

7/7/36 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

14221068 PMID: 9973501

TCR vaccines against T cell lymphoma: QS-21 and IL-12 adjuvants induce a protective CD8+ T cell response.

Wong C P; Okada C Y; Levy R
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Journal of immunology (Baltimore, Md. - 1950) (UNITED STATES) Feb 15 1999, 162 (4) p2251-8, ISSN 0022-1767 Journal Code: 2985117R
Contract/Grant No.: CA 69521; CA; NCI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Tumor-specific TCR can serve as an effective target for active immunotherapy of T cell malignancies. Using the murine T cell tumor model C6VL, vaccination with C6VL TCR protected mice from a subsequent lethal dose of tumor cells. This study characterizes the immune mechanisms involved in the tumor protection, and the influence of immunologic

adjuvants in ***inducing*** a protective immune response. Immune responses **induced** by **TCR** vaccines formulated with various adjuvants: QS-21, IL-12, SAF-1, ***CD40L*** , and GM-CSF were compared. QS-21, IL-12, and SAF-1 biased the humoral immune response toward Th1-type, reflected by the ***induction*** of IgG2a and IgG2b anti-C6VL ***TCR*** Abs. ***CD40L*** and GM-CSF exclusively produced IgG1 Abs, reflecting a Th2-type immune response. In our tumor model system, only vaccines containing adjuvants that induced a Th1-type immune response favored tumor protection. Furthermore, we demonstrated that CD8+ T cells were necessary and sufficient for tumor protection using anti-CD8 mAb depletion and adoptive cell transfer experiments. Transfer of hyperimmune serum containing anti-C6VL TCR Abs into na ive mice had modest anti-tumor effects and was not sufficient to prevent tumor growth. TCR-vaccinated B cell-deficient mice were not protected against C6VL tumor, and tumor protection was not completely restored after hyperimmune serum transfer. Thus, B cells may serve as important APCs in ***inducing*** a protective immune response. Based on these results future **TCR** vaccines should be designed to maintain native **TCR** conformation, as well as **induce** a strong Th1-type immune response.

Record Date Created: 19990413

Record Date Completed: 19990413

7/7/37 (Item 5 from file: 155)

DIALOG(R)File 155:MEDLINE(R)

14220960 PMID: 9973393

CD40-CD154 interaction and IFN-gamma are required for IL-12 but not prostaglandin E2 secretion by microglia during antigen presentation to Th1 cells.

Aloisi F; Penna G; Polazzi E; Minghetti L; Adorini L

Laboratory of Organ and System Pathophysiology, Istituto Superiore di Sanita, Rome, Italy.

Journal of immunology (Baltimore, Md. - 1950) (UNITED STATES) Feb 1 1999, 162 (3) p1384-91, ISSN 0022-1767 Journal Code: 2985117R

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

IL-12 and PGE2 promote and inhibit, respectively, the development of Th1 responses. Production of these mediators by APC residing in the central nervous system (CNS) may be involved in the local regulation of the T cell phenotype during infectious and autoimmune CNS diseases. In the present study we have examined IL-12 and PGE2 secretion by cultured microglia and astrocytes from the mouse brain upon Ag-dependent interaction with I-Ad-restricted, OVA323-339 specific TCR transgenic Th1 and Th2 cell lines. We show that microglia, which restimulate efficiently both Th1 and Th2 cells, secrete IL-12 upon Ag-dependent interaction with Th1, but not with Th2 cells. Th1-driven IL-12 production depends on ***TCR*** ligation by MHC class II/peptide complexes, **CD40** engagement on microglia, and IFN-gamma secretion by activated Th1 cells. Th1 and, to a lesser extent, Th2 cells also stimulate the production of PGE2 by microglia. T cell-mediated **induction** of PGE2 requires MHC class II/peptide/ **TCR** interactions but does not depend on **CD40** engagement or on the presence of IFN-gamma. Astrocytes, which preferentially activate Th2 cells, fail to produce IL-12 and secrete negligible amounts of PGE2 upon interaction with either Th1 or Th2 cells. These results suggest that during CNS infection or immunopathology, IL-12 produced by microglia upon Ag-specific interaction with Th1 cells may further skew the immune response to Th1, whereas the T cell-dependent production of PGE2 by microglia may represent a negative feedback mechanism, limiting the propagation of Th1 responses.

Record Date Created: 19990413
Record Date Completed: 19990413

7/7/38 (Item 6 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

14042592 PMID: 9743383
Signaling through a CD3 gamma-deficient TCR/CD3 complex in immortalized mature CD4+ and CD8+ T lymphocytes.
Pacheco-Castro A; Alvarez-Zapata D; Serrano-Torres P; Regueiro J R
Immunologia, Facultad de Medicina, Universidad Complutense, Madrid, Spain.
Journal of immunology (Baltimore, Md. - 1950) (UNITED STATES) Sep 15 1998, 161 (6) p3152-60, ISSN 0022-1767 Journal Code: 2985117R
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
The biologic role of each CD3 chain and their relative contribution to the signals transduced through the TCR/CD3 complex and to downstream activation events are still controversial: they may be specialized or redundant. We have immortalized peripheral blood CD4+ and CD8+ T lymphocytes from a human selective CD3 gamma deficiency using Herpesvirus saimiri. The accessibility of the mutant TCR/CD3 complex to different Abs was consistently lower in immortalized CD8+ cells when compared with CD4+ cells, relative to their corresponding CD3 gamma-sufficient controls. Several **TCR**/CD3-**induced** downstream activation events, immediate (calcium flux), early (cytotoxicity and induction of surface CD69 or **CD40L** activation markers or intracellular TNF-alpha) and late (proliferation and secretion of TNF-alpha), were normal in gamma-deficient cells, despite the fact that their TCR/CD3 complexes were significantly less accessible than those of controls. In contrast, the accumulation of intracellular IL-2 or its secretion after CD3 triggering was severely impaired in gamma-deficient cells. The defect was upstream of protein kinase C activation because addition of transmembrane stimuli (PMA plus calcium ionophore) completely restored IL-2 secretion in gamma-deficient cells. These results suggest that the propagation of signals initiated at the TCR itself can result in a modified downstream signaling cascade with distinct functional consequences when gamma is absent. They also provide evidence for the specific participation of the CD3 gamma chain in the induction of certain cytokine genes in both CD4+ and CD8+ human mature T cells. These immortalized mutant cells may prove to be useful in isolating cytosolic signaling pathways emanating from the TCR/CD3 complex.
Record Date Created: 19981006
Record Date Completed: 19981006

7/7/39 (Item 7 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13028674 PMID: 8724831
Regulation of T lymphocyte subsets.
Fitch F W; Stack R; Fields P; Lancki D W; Cronin D C
Department of Pathology, University of Chicago, IL 60637, USA.
Ciba Foundation symposium (NETHERLANDS) 1995, 195 p68-80; discussion 80-5, ISSN 0300-5208 Journal Code: 0356636
Contract/Grant No.: AI-29531; AI; NIAID; AI-35294; AI; NIAID; CA-44372; CA; NCI
Document type: Journal Article; Review; Review, Tutorial
Languages: ENGLISH
Main Citation Owner: NLM

Record type: Completed
Patterns of cytokine secretion and functional differences distinguish T lymphocyte subsets. T lymphocyte subsets are also regulated differentially. Most established CD8+ lymphocyte clones secrete gamma-interferon (IFN-gamma) but not interleukin 2 (IL-2) or IL-4. Using murine T cells which express a transgenic, antigen-specific alpha/beta T cell receptor (TCR) specific for L(d) class I major histocompatibility complex antigen, we have found that CD8+ lymphocytes can be divided into functional subsets. Freshly isolated CD8+ T cells are not cytolytic, do not proliferate and do not proliferate and do not secrete cytokines. Stimulation of ***TCR*** alone does not **induce** cytokine secretion, but cells become responsive to exogenous IL-2 or IL-4. Stimulation of CD28 together with **TCR** **induces** secretion of IL-2 and IFN-gamma, and cells proliferate without exogenous cytokines. Proliferation is necessary for the development of cytolytic activity. If IL-4 is present during initial stimulation, IL-4 is secreted following restimulation. Upon stimulation, some IL-4-producing murine CD8+ T cell clones express CD40 ligand (CD40L), and they potentiate proliferation and immunoglobulin secretion by small resting B cells. Thus, the CD8+ T cell subsets T cytotoxic 1 (Tc1) and Tc2 are analogous to CD4+ T helper 1 (Th1) and Th2. IL-2 production by naive CD8+ cells requires co-stimulation. IL-4 production by CD8+ T cells requires the presence of IL-4 during initial stimulation. Some IL-4-producing CD8+ T cells express **CD40L** following **TCR** stimulation and provide help for B cells. (42 Refs.)
Record Date Created: 19960916
Record Date Completed: 19960916

7/7/40 (Item 8 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

12630788 PMID: 7751632
Regulation of early T cell development by the engagement of TCR-beta complex expressed on fetal thymocytes from TCR-beta-transgenic scid mice.
Takahama Y; Sugaya K; Tsuda S; Hasegawa T; Hashimoto Y
Institute of Immunology, Syntex, Ibaraki, Japan.
Journal of immunology (Baltimore, Md. - 1950) (UNITED STATES) Jun 1 1995, 154 (11) p5862-9, ISSN 0022-1767 Journal Code: 2985117R
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Transgenic expression of the beta-chain of T cell antigen-receptor (**TCR**) is known to **induce** the generation of CD4+ CD8+ thymocytes in the immunodeficient scid mouse, in which thymocyte development is otherwise arrested at CD4- CD8- cells. It is not clear, however, whether or not the thymocyte development is controlled by ligand engagement of the TCR-beta complex on the cell surface. In the present study, we have examined how the engagement by Ab of the TCR-beta complex expressed on the TCR-beta-transgenic scid fetal thymocytes can regulate the generation of CD4+ CD8+ thymocytes. Organ cultures of CD4- CD8- day 14 fetal thymocytes from the **TCR**-beta-transgenic scid mice resulted in the generation of CD4- CD8+ and then CD4+ CD8+ cells. The initial step from ***CD40*** - CD8- cells to CD4- CD8+ cells was enhanced by the addition of anti-**TCR** -beta Ab, whereas the subsequent step from CD4- CD8+ cells to CD4+ CD8+ cells was markedly inhibited by anti-TCR-beta Ab. These results indicate that ligand engagement of the TCR-beta complex can positively and negatively regulate the early thymocyte development. Moreover, the finding that engagement of TCR-beta complex inhibits the generation of CD4+ CD8+ cells suggests that the **induction** of CD4+ CD8+ thymocytes by the **TCR** -beta transgene is not an immediate consequence of cell-surface engagement of the TCR-beta complex but requires liberation from the continued TCR-beta signaling.

Record Date Created: 19950622
Record Date Completed: 19950622

7/7/41 (Item 9 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

11690780 PMID: 11865410
Temporal sequence and functional implications of V beta-specific T cell receptor down-regulation and costimulatory molecule expression following in vitro stimulation with the staphylococcal superantigen Toxic shock syndrome toxin-1.
Kum Winnie W S; Hung Ryan W Y; Cameron Scott B; Chow Anthony W
Department of Medicine, Division of Infectious Diseases, University of British Columbia, Vancouver Hospital, Vancouver, Canada.
Journal of infectious diseases (United States) Feb 15 2002, 185 (4) p555-60, ISSN 0022-1899 Journal Code: 0413675
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
The superantigen toxic shock syndrome toxin-1 (TSST-1) is implicated as the major cause of staphylococcal toxic shock syndrome. The temporal sequence of early signaling events in human peripheral blood mononuclear cells following TSST-1 stimulation was examined. TSST-1 ***induced*** rapid and complete down-regulation of V beta 2-specific **T cell receptor** (**TCR**), followed by transient CD154 expression on CD4(+) lymphocytes. This was sequentially followed by the up-regulation of CD86, CD80, **CD40** , and human leukocyte antigen-DR expression on CD14(+) monocytes. In contrast, S14N, a TSST-1 mutant toxin with a single amino acid substitution that is known to be impaired in interleukin (IL)--2, interferon (IFN)-gamma, and tumor necrosis factor (TNF)-alpha secretion, was deficient in both V beta 2-TCR down-regulation and CD154 and CD80/CD86 expression. Furthermore, pretreatment with monoclonal antibodies against V beta 2-**TCR**, CD80/CD86, and CD154 significantly inhibited TSST-1- ***induced*** IL-2, IFN-gamma, and TNF-alpha secretion. Taken together, these results indicate that early V beta-specific TCR activation, along with CD80/CD86 and CD154 costimulation, are key determinants of the TSST-1-induced proinflammatory cytokine response.
Record Date Created: 20020226
Record Date Completed: 20020315
Date of Electronic Publication: 20020122

7/7/42 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

136261819 CA: 136(17)261819j PATENT
Coupling of peripheral tolerance to endogenous IL-10 promotes effective modulation of T cells and ameliorates autoimmune disease
INVENTOR(AUTHOR): Zaghouani, Habib
LOCATION: USA
PATENT: U.S. Pat. Appl. Publ. ; US 20020038002 A1 DATE: 20020328
APPLICATION: US 873901 (20010604) *US PV209527 (20000605)
PAGES: 84 pp. CODEN: USXXCO LANGUAGE: English CLASS: 530388220; A61K-039/395A; C07K-016/28B
SECTION:
CA215002 Immunochemistry
IDENTIFIERS: immunomodulator Fc receptor ligand immunosuppressive antigen , autoimmune disease tolerance chimeric Ig autoantigen
DESCRIPTORS:
T cell(lymphocyte)...

activation, inhibition; compns. comprising tolerance-inducing autoantigenic peptide and Fc receptor ligand or TCR antagonist or agonist for ameliorating autoimmune diseases
Immunostimulants...
adjuvants, non-; compns. comprising tolerance-inducing autoantigenic peptide and Fc receptor ligand or TCR antagonist or agonist for ameliorating autoimmune diseases
TCR(T cell receptors)...
agonist or antagonist; compns. comprising tolerance-inducing autoantigenic peptide and Fc receptor ligand or TCR antagonist or agonist for ameliorating autoimmune diseases
Cytokines...
anti-inflammatory; compns. comprising tolerance-inducing autoantigenic peptide and Fc receptor ligand or TCR antagonist or agonist for ameliorating autoimmune diseases
Antigens...
autoantigens; compns. comprising tolerance-inducing autoantigenic peptide and Fc receptor ligand or TCR antagonist or agonist for ameliorating autoimmune diseases
Transforming growth factors...
β-, prodn. promotion; compns. comprising tolerance-inducing autoantigenic peptide and Fc receptor ligand or TCR antagonist or agonist for ameliorating autoimmune diseases
Drug delivery systems...
carriers; compns. comprising tolerance-inducing autoantigenic peptide and Fc receptor ligand or TCR antagonist or agonist for ameliorating autoimmune diseases
Immunity...
cell-mediated; compns. comprising tolerance-inducing autoantigenic peptide and Fc receptor ligand or TCR antagonist or agonist for ameliorating autoimmune diseases
Proteolipid protein... Myelin basic protein...
chimeric; compns. comprising tolerance-inducing autoantigenic peptide and Fc receptor ligand or TCR antagonist or agonist for ameliorating autoimmune diseases
Immunomodulators... Immunoglobulin receptors... Ligands...
Immunosuppressants... Antigens... Fusion proteins(chimeric proteins)...
Antibodies... Immunoglobulins... Allergy... Transplant rejection...
Autoimmune disease... Rheumatoid arthritis... Multiple sclerosis...
Vertebrata... Newborn... Allergens... Antigen-presenting cell...
Combinatorial chemistry... Physiological saline solutions... Microspheres ... Microparticles... Latex... Lipids,biological studies...
Albumins,biological studies... Polymers,biological studies...
Precipitation(chemical)... Protein sequences... Lupus erythematosus...
compns. comprising tolerance-inducing autoantigenic peptide and Fc receptor ligand or TCR antagonist or agonist for ameliorating autoimmune diseases
Immunity...
disorder; compns. comprising tolerance-inducing autoantigenic peptide and Fc receptor ligand or TCR antagonist or agonist for ameliorating autoimmune diseases
Immunoglobulins...
fragments; compns. comprising tolerance-inducing autoantigenic peptide and Fc receptor ligand or TCR antagonist or agonist for ameliorating autoimmune diseases
Immunoglobulins...
G; compns. comprising tolerance-inducing autoantigenic peptide and Fc receptor ligand or TCR antagonist or agonist for ameliorating autoimmune diseases
Interferons...
γ, prodn. inhibition; compns. comprising tolerance-inducing autoantigenic peptide and Fc receptor ligand or TCR antagonist or agonist for ameliorating autoimmune diseases
Absorbents... Aggregates...

Ig.; compns. comprising tolerance-inducing autoantigenic peptide and Fc receptor ligand or TCR antagonist or agonist for ameliorating autoimmune diseases
Development,mammalian postnatal...
infant; compns. comprising tolerance-inducing autoantigenic peptide and Fc receptor ligand or TCR antagonist or agonist for ameliorating autoimmune diseases
CD80(antigen)... CD86(antigen)... CD40(antigen)...
inhibition; compns. comprising tolerance-inducing autoantigenic peptide and Fc receptor ligand or TCR antagonist or agonist for ameliorating autoimmune diseases
Diabetes mellitus...
insulin-dependent; compns. comprising tolerance-inducing autoantigenic peptide and Fc receptor ligand or TCR antagonist or agonist for ameliorating autoimmune diseases
Histocompatibility antigens...
MHC (major histocompatibility complex), class II; compns. comprising tolerance-inducing autoantigenic peptide and Fc receptor ligand or TCR antagonist or agonist for ameliorating autoimmune diseases
Evolution...
mol., directed; compns. comprising tolerance-inducing autoantigenic peptide and Fc receptor ligand or TCR antagonist or agonist for ameliorating autoimmune diseases
Interleukin 10... Interleukin 6... Interleukin 4... Interleukin 9...
Interleukin 13... Interleukins...
prodn. promotion; compns. comprising tolerance-inducing autoantigenic peptide and Fc receptor ligand or TCR antagonist or agonist for ameliorating autoimmune diseases
Drug design...
rational; compns. comprising tolerance-inducing autoantigenic peptide and Fc receptor ligand or TCR antagonist or agonist for ameliorating autoimmune diseases
Connective tissue...
scleroderma; compns. comprising tolerance-inducing autoantigenic peptide and Fc receptor ligand or TCR antagonist or agonist for ameliorating autoimmune diseases
Cell activation...
T cell, inhibition; compns. comprising tolerance-inducing autoantigenic peptide and Fc receptor ligand or TCR antagonist or agonist for ameliorating autoimmune diseases
Immune tolerance...
T cell; compns. comprising tolerance-inducing autoantigenic peptide and Fc receptor ligand or TCR antagonist or agonist for ameliorating autoimmune diseases
T cell(lymphocyte)...
tolerance; compns. comprising tolerance-inducing autoantigenic peptide and Fc receptor ligand or TCR antagonist or agonist for ameliorating autoimmune diseases
Intestine,disease...
ulcerative colitis; compns. comprising tolerance-inducing autoantigenic peptide and Fc receptor ligand or TCR antagonist or agonist for ameliorating autoimmune diseases
CAS REGISTRY NUMBERS:
7783-20-2 biological studies, compns. comprising tolerance-inducing autoantigenic peptide and Fc receptor ligand or TCR antagonist or agonist for ameliorating autoimmune diseases
18358-13-9 biological studies, microparticles; compns. comprising tolerance-inducing autoantigenic peptide and Fc receptor ligand or TCR antagonist or agonist for ameliorating autoimmune diseases
210418-48-7P 210418-49-8P 172228-98-7P chimeric; compns. comprising tolerance-inducing autoantigenic peptide and Fc receptor ligand or TCR antagonist or agonist for ameliorating autoimmune diseases
9003-39-8 compns. comprising tolerance-inducing autoantigenic peptide and Fc receptor ligand or TCR antagonist or agonist for ameliorating

autoimmune diseases

7/7/43 (Item 2 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

132077527 CA: 132(7)77527d JOURNAL
Essential role for both CD80 and CD86 costimulation, but not CD40 interactions, in allergen-induced Th2 cytokine production from asthmatic bronchial tissue: role for αβ, but not γδ, T cells
AUTHOR(S): Jaffar, Zeina H.; Stanciu, Luminita; Pandit, Anita; Lordan, James; Holgate, Stephen T.; Roberts, Kevan
LOCATION: Southampton General Hospital, University Medicine, Southampton, UK, SO16 6YD
JOURNAL: J. Immunol. DATE: 1999 VOLUME: 163 NUMBER: 11 PAGES: 6283-6291 CODEN: JOIMA3 ISSN: 0022-1767 LANGUAGE: English PUBLISHER: American Association of Immunologists
SECTION:
CA215009 Immunochemistry
IDENTIFIERS: CD80 CD86 costimulation allergen specific TCRalphabeta T cell asthma
DESCRIPTORS:
CD40(antigen)... CD80(antigen)... CD86(antigen)... Interleukin 13... Interleukin 4... Interleukin 5...
allergen-specific TCRαβ T cells but not TCRγδ T cells are resident in asthmatic tissue and costimulation by both CD80 and CD86 but not CD40/CD40L is essential for allergen-induc
Asthma...
allergic; allergen-specific TCRαβ T cells but not TCRγδ T cells are resident in asthmatic tissue and costimulation by both CD80 and CD86 but not CD40/CD40L is essential for alle
Glycoproteins,specific or class...
CD40-L (antigen CD40 ligand); allergen-specific TCRαβ T cells but not TCRγδ T cells are resident in asthmatic tissue and costimulation by both CD80 and CD86 but not CD40/CD40L i
T cell(lymphocyte)...
helper cell/inducer, TH2; allergen-specific TCRαβ T cells but not TCRγδ T cells are resident in asthmatic tissue and costimulation by both CD80 and CD86 but not CD40/CD40L is es
T cell(lymphocyte)...
TCRαβ-pos.; allergen-specific TCRαβ T cells but not TCRγδ T cells are resident in asthmatic tissue and costimulation by both CD80 and CD86 but not CD40/CD40L is essen
T cell(lymphocyte)...
TCRγδ-pos.; allergen-specific TCRαβ T cells but not TCRγδ T cells are resident in asthmatic tissue and costimulation by both CD80 and CD86 but not CD40/CD40L is esse

7/7/44 (Item 3 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

131086583 CA: 131(7)86583a JOURNAL
A Role for CD99 in T Cell Activation
AUTHOR(S): Wingett, Denise; Forcier, Kristin; Nielson, Christopher P.
LOCATION: Department of Veterans Affairs Medical Center, Boise, ID, 83702, USA
JOURNAL: Cell. Immunol. DATE: 1999 VOLUME: 193 NUMBER: 1 PAGES: 17-23
CODEN: CLIMB8 ISSN: 0008-8749 LANGUAGE: English PUBLISHER: Academic Press
SECTION:

CA215002 Immunochemistry
IDENTIFIERS: CD99 costimulatory signal T cell activation
DESCRIPTORS:
Interleukin 2 receptors...
α-chain; CD99 signal transduction delivers costimulatory signals in CD4-pos. human T cell activation induced by TCR receptor
TCR(T cell receptors)...
CD3 complex; CD99 signal transduction delivers costimulatory signals in CD4-pos. human T cell activation induced by TCR receptor
Glycoproteins,specific or class...
CD40-L (antigen CD40 ligand); CD99 signal transduction delivers costimulatory signals in CD4-pos. human T cell activation induced by TCR receptor
CD4-positive T cell... CD69(antigen)...
CD99 signal transduction delivers costimulatory signals in CD4-pos. human T cell activation induced by TCR receptor
CD antigens...
CD99; CD99 signal transduction delivers costimulatory signals in CD4-pos. human T cell activation induced by TCR receptor
Cell activation... Cell proliferation...
T-cell; CD99 signal transduction delivers costimulatory signals in CD4-pos. human T cell activation induced by TCR receptor
CD3(antigen)...
TCR complex; CD99 signal transduction delivers costimulatory signals in CD4-pos. human T cell activation induced by TCR receptor

7/7/45 (Item 4 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

129134960 CA: 129(11)134960d JOURNAL
A humanized therapeutic CD4 mAb inhibits TCR-induced IL-2, IL-4, and IL-10 secretion and expression of CD25, CD40L, and CD69
AUTHOR(S): Woods, Margaret; Guy, Robert; Waldmann, Herman; Glennie, Martin; Alexander, Denis R.
LOCATION: T Cell Laboratory, Department of Immunology, Babraham Inst., Cambridge, UK, CB2 4AT
JOURNAL: Cell. Immunol. DATE: 1998 VOLUME: 185 NUMBER: 2 PAGES: 101-113 CODEN: CLIMB8 ISSN: 0008-8749 LANGUAGE: English PUBLISHER: Academic Press
SECTION:
CA215005 Immunochemistry
IDENTIFIERS: CD4 antibody T lymphocyte activation inflammation
DESCRIPTORS:
Monoclonal IgG1...
anti CD4 antigen; humanized therapeutic CD4 mAb inhibits TCR-induced IL-2, IL-4, and IL-10 secretion and expression of CD25, CD40L, and CD69
CD40 ligand... CD4(antigen)... CD4-positive T cell... CD69(antigen)... Cell cycle... Immunosuppressants... Inflammation... Interleukin 10... Interleukin 2 receptors... Interleukin 2... Interleukin 4... Signal transduction(biological)... T cell activation... T cell proliferation... TCR(T cell receptors)...
humanized therapeutic CD4 mAb inhibits TCR-induced IL-2, IL-4, and IL-10 secretion and expression of CD25, CD40L, and CD69

7/7/46 (Item 5 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

128216369 CA: 128(18)216369m PATENT
Bi- and trispecific antibodies for induction of tumor immunity
INVENTOR(AUTHOR): Lindhofer, Horst; Kolb, Hans-Jochem; Thierfelder,

Stefan
LOCATION: Germany,
ASSIGNEE: GSF-Forschungszentrum fuer Umwelt und Gesundheit G.m.b.H. Neuherberg
PATENT: Germany Offen. ; DE 19710497 A1 DATE: 19980305
APPLICATION: DE 19710497 (19970313) *DE 19635743 (19960903) *DE 19648976 (19961126)
PAGES: 18 pp. CODEN: GWXXBX LANGUAGE: German CLASS: C07K-016/28A; A61K-039/395B
SECTION:
CA215003 Immunochemistry
IDENTIFIERS: tumor immunotherapy antibody TCR FcR antigen
DESCRIPTORS:
Anergy... Antitumor agents... CD28(antigen)... CD2(antigen)... CD3(antigen) ... CD40(antigen)... CD44(antigen)... CD5(antigen)... CD80(antigen)... CD86(antigen)... Class I MHC antigens... Class II MHC antigens... Complement... Cytokines... Dendritic cell... Fc receptors... FcγRI receptors... FcγRII receptors... FcγRIII receptors... Humoral immunity... ICAM-1(cell adhesion molecule)... IgG1... IgG2a... IgG2b... IgG3... IgG4... Immunotherapy... Interleukin 12... Interleukin 1... Interleukin 2... Interleukin 4... Interleukin 6... Interleukin 8... LFA-3(antigen)... Macrophage... Monocyte... Mouse... Rat... T cell proliferation... T cell(lymphocyte)... TCR(T cell receptors)... Tumor necrosis factor α... Tumors(animal)... Tumor-associated antigen...
bispecific and trispecific antibodies to TCR receptor complex, tumor-assocd. antigens and Fc receptors for induction of tumor immunity
Antibodies...
bispecific; bispecific and trispecific antibodies to TCR receptor complex, tumor-assocd. antigens and Fc receptors for induction of tumor immunity
Antigens...
costimulatory; bispecific and trispecific antibodies to TCR receptor complex, tumor-assocd. antigens and Fc receptors for induction of tumor immunity
Antibodies...
trispecific; bispecific and trispecific antibodies to TCR receptor complex, tumor-assocd. antigens and Fc receptors for induction of tumor immunity
Peptides,biological studies...
tumor-specific; bispecific and trispecific antibodies to TCR receptor complex, tumor-assocd. antigens and Fc receptors for induction of tumor immunity

7/7/47 (Item 6 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

122237721 CA: 122(19)237721m JOURNAL
A subset of CD4+ memory T cells contains preformed CD40 ligand that is rapidly but transiently expressed on their surface after activation through the T cell receptor complex
AUTHOR(S): Palleja, Casamayor, Montserrat; Khan, Mahmood; MacLennan, Ian C. M.
LOCATION: Dep. Immunology, Univ. Birmingham Med. Sch., Birmingham, UK, B15 2TT
JOURNAL: J. Exp. Med. DATE: 1995 VOLUME: 181 NUMBER: 4 PAGES: 1293-301 CODEN: JEMEAV ISSN: 0022-1007 LANGUAGE: English
SECTION:
CA215010 Immunochemistry
IDENTIFIERS: CD40 ligand T lymphocyte TCR receptor
DESCRIPTORS:
Antigen receptors,TCR (T-cell antigen receptor)... Glycoproteins,specific or class, CD40-L (antigen CD40 ligand)... Lymphocyte,B-cell...

Lymphocyte,T-cell... Receptors,TCR (T-cell antigen receptor)... Signal transduction,biological... Tonsil...

CD40 ligand expression on human tonsil T cells can be induced by signaling through TCR complexes

?